THE EFFECT OF KETAMINE ON THE ONSET TIME AND THE INTUBATING CONDITIONS OF ROCURONIUM BROMIDE

Dissertation submitted to

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CERTIFICATE

This is to certify that the dissertation entitled, **"THE EFFECT OF KETAMINE ON THE ONSET TIME AND THE INTUBATING CONDITIONS OF ROCURONIUM BROMIDE",** Submitted by Dr. NAVEEN KUMAR.D in partial fulfilment for the award of the degree of Doctor of Medicine in Anaesthesiology by the Tamilnadu Dr. M.G.R. Medical University, Chennai is a bonafide record of the work done by him in the INSTITUTE OF ANAESTHESIOLOGY & CRITICAL CARE, Madras Medical College, during the academic year 2011-2013.

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DECLARATION

I, **Dr. NAVEEN KUMAR. D** solemnly declare that this dissertation entitled **"THE EFFECT OF KETAMINE ON THE ONSET TIME AND THE INTUBATING CONDITIONS OF ROCURONIUM BROMIDE"** is a bonafide work done by me in the Institute of Anaesthesiology & critical care, Madras Medical College and Rajiv Gandhi Government General hospital, Chennai, during the period 2010-2013 under the able guidance of **Prof. *M. VASANTHI, MD., DA., DNB.,*** Director, Institute of anaesthesiology & critical care, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai – 3 and submitted to **The Tamilnadu Dr. MGR Medical University,** Guindy, Chennai – 32, in the partial fulfillment of the requirements for the award of the degree of MD Anaesthesiology (Branch X).

Place:

Date:

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CONTENTS

INTRODUCTION

General anaesthesia can be administered by multiple means to a patient. Most common method used is with the help of endotracheal intubation and artificially controlling a patient's ventilation.

The occurrence of major complications such as aspiration and hypoxia, especially in patients coming for emergency surgery, obese patients or cesarean section where rapid sequence induction is required, depends on how fast an anesthesiologist is able to secure the airway. This in turn depends on the onset time of proper intubating conditions.

Satisfactory intubating conditions can be obtained by using multitude of drugs and methods. Most common method is to use an induction agent along with administration of suxamethonium for muscle relaxation. Even though suxamethonium produces excellent intubating conditions with a very short onset time, it has its own limitations.

It is contraindicated or better avoided in patients with hyperkalemia, pseudocholinesterase deficiency, burns, penetrating eye injury, allergic reactions, increased intracranial pressure. Also, Suxamethonium induced muscle relaxation produces myalgia postoperatively.

Rocuronium has in the recent past emerged as a useful alternative to suxamethonium for rapid sequence induction especially when suxamethonium is contraindicated. It is an aminosteroid derivative with very short onset of action. To use it for Rapid sequence induction it has to be used at thrice its ED_{95} dosage which leads to prolonged duration of action.

Various strategies were devised to use a lower dose for RSI thereby limiting its duration of action. Priming was used in many studies without any conclusive results either for or against the technique. Another method is to reduce the effect site equilibrium time, shortening it will lead to a faster onset. This has been extensively studied using recirculatory pharmacokinetic models. Using indocyanine green as a marker it has been proved that cardiac output influences the pharmacokinetics of Rocuronium.

Many drugs have been given before induction to maintain or improve the cardiac output. Ephedrine has been used previously to increase the cardiac output and reduce the onset time of Rocuronium by as much as 26%. Intubating conditions were also better with the use of Ephedrine. Esmolol, a beta blocker, which reduces the cardiac output, was found to prolong the onset time.

Ketamine, a NMDA antagonist, has a sympathomimetic property. It has been used extensively as an induction agent in cases of hypovolemic shock. It maintains the cardiac output. In addition it has anesthetic properties which will help in providing better intubating conditions.

Thus, we hypothesized that addition of low dose Ketamine to propofol Rocuronium induction will shorten the onset time and produce better intubating conditions.

AIM OF THE STUDY

The aim of the study was to evaluate the effects of Ketamine and normal saline given before induction on the onset time and intubating conditions of Rocuronium bromide

The parameters to be evaluated are

- The onset time of Rocuronium
- The intubating conditions
- Cormack lehanne grading
- Percentage of glottis opening

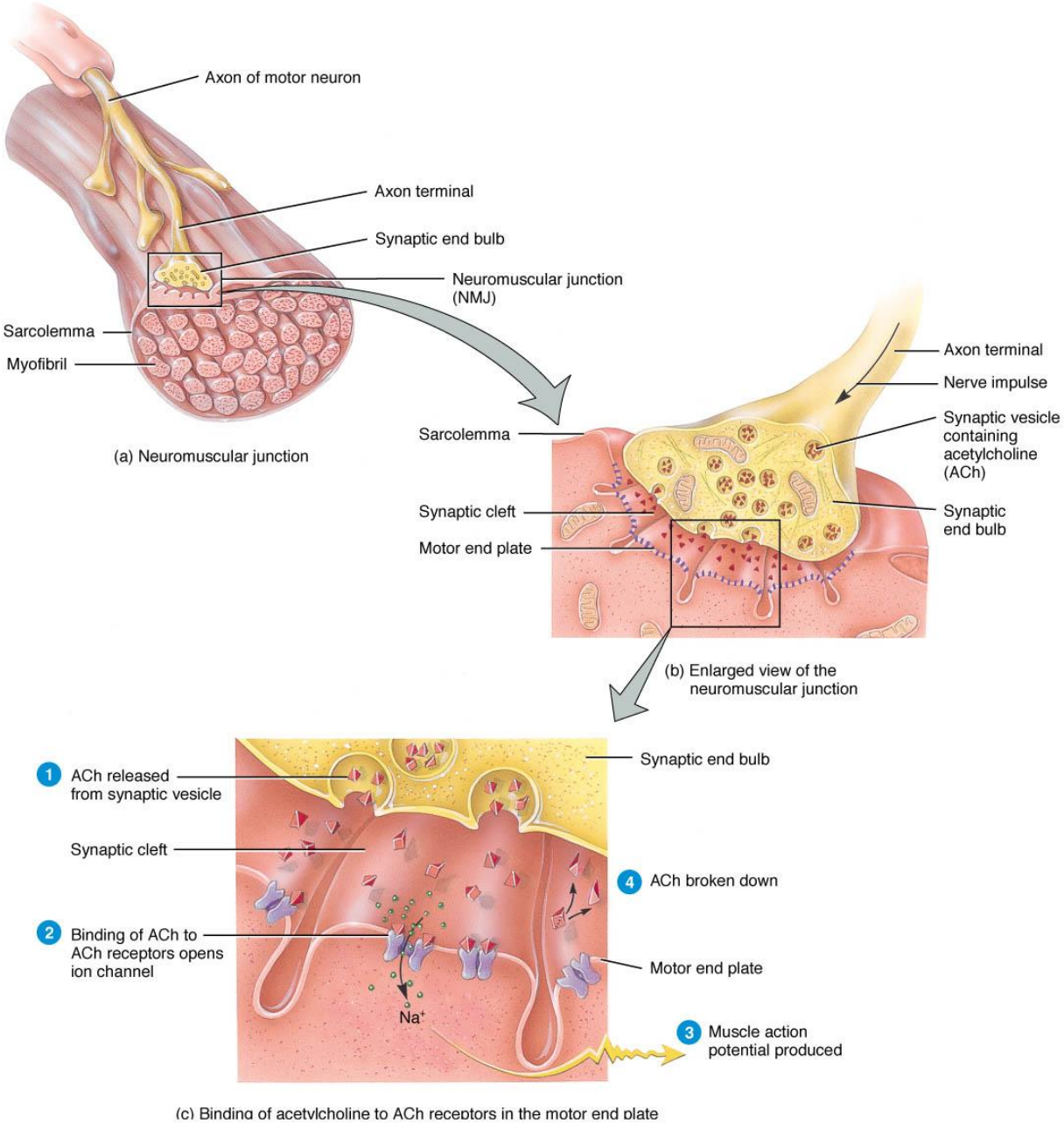
The secondary outcomes to be measured are

- Heart rate variations
- Hemodynamic alterations during the study

FUNCTIONAL ANATOMY OF NEUROMUSCULAR JUNCTION

Neuromuscular junction is a specialized structure at the interface of nerve and muscle where the messages from the motor cortex are transmitted through chemical mediators. From the anterior horn of spinal cord and the medulla the motor neuron travels as a thick myelinated axon. In the muscles, it branches out to form contact with muscle to form many functional parts known as motor unit. Stimulation of a neuron causes all the muscle cells in the motor unit to contract synchronously to cause fasciculations.

The nerve and muscle are separated by a slender distance of 20 nm. This portion is called synaptic cleft. The muscular surface is corrugated into primary and secondary clefts, thereby increasing the surface area. Acetylcholine receptors are situated at the shoulder of the cleft while the sodium channels are located deep within.



Perijunctional area is rich in sodium channels and here aceylcholine receptors are less dense. The vesicles containing acetylcholine are located near electron dense thickened portion of membrane known as active site. Mitochondria, microtubules and other support structures are located on its opposite side.

The end plate potential is the sum total of miniature end plate potentials caused by release of small vesicles from the end plate. Release of vesicles usually occurs after a stimulus. Each stimulus releases 200 quanta. Each quanta contains 5000 acetylcholine molecules which can stimulate around five lakh molecules of acetylcholine receptors. Transmission across the junction has substantial margin for safety and at the same time has a lot of reserve.

STEPS OF TRANSMISSION:

1. Acetate and choline from the nerve ending form the substrate. They are transported into the cell where acetate is converted to aceyl CoA in mitochondria. Acetyl Coa in the presence of choline acetyl transferase binds with choline to form acetylcholine.

2. This acetylcholine thus formed is stored in cytoplasm. Once required they are packed into vesicles and transported to the end-plate for release.

3. Once a nerve is stimulated sodium enters the cell causing depolarisation and release of calcium into the neurons. This causes the release of acetylcholine into the synaptic cleft. Calcium plays

an important role in nerve terminal. Accumulation of calcium can increase the force of contraction and can overcome neuromuscular blockade. This is classically seen in post-tetanic contraction.

4. Other conditions such as eaten lambert syndrome and myasthenia gravis, where there is antibody against calcium channels, classically produce weakness. Magnesium and other divalent cations can compete with calcium and can prolong neuromuscular blockade.

5. Synaptic release and recycling : the nerve terminal contains two type of vesicles VP1 (reserve vesicles) and VP2 (releasable pool).they are tethered to the membrane by proteins such as synaptobrevin, synphysin and others. binding of calcium to the SNARE proteins causes these vesicles to fuse with the membrane and release its contents.

Toxins from botulinum and tetanus block the release of vesicles by damaging one of the SNARE proteins. Since the release of acetylcholine is inhibited it leads to paralysis

6. Once acetylcholine is released it into the cleft, it reacts with receptors in the muscle end to initiate contraction.

7. The acetylcholine that is not bound to the receptors, once it is released into the synapse, is metabolised immediately by acetylcholine esterase. Acetylcholine molecule is one of the most potent messengers in human body but its action is terminated in milliseconds due to the efficient activity of acetylcholine esterase.

Post junctional acetylcholine receptors are of three types. They are

- Junctional or mature
- Extrajunctional or immature
- Neuronal α 7 receptors.

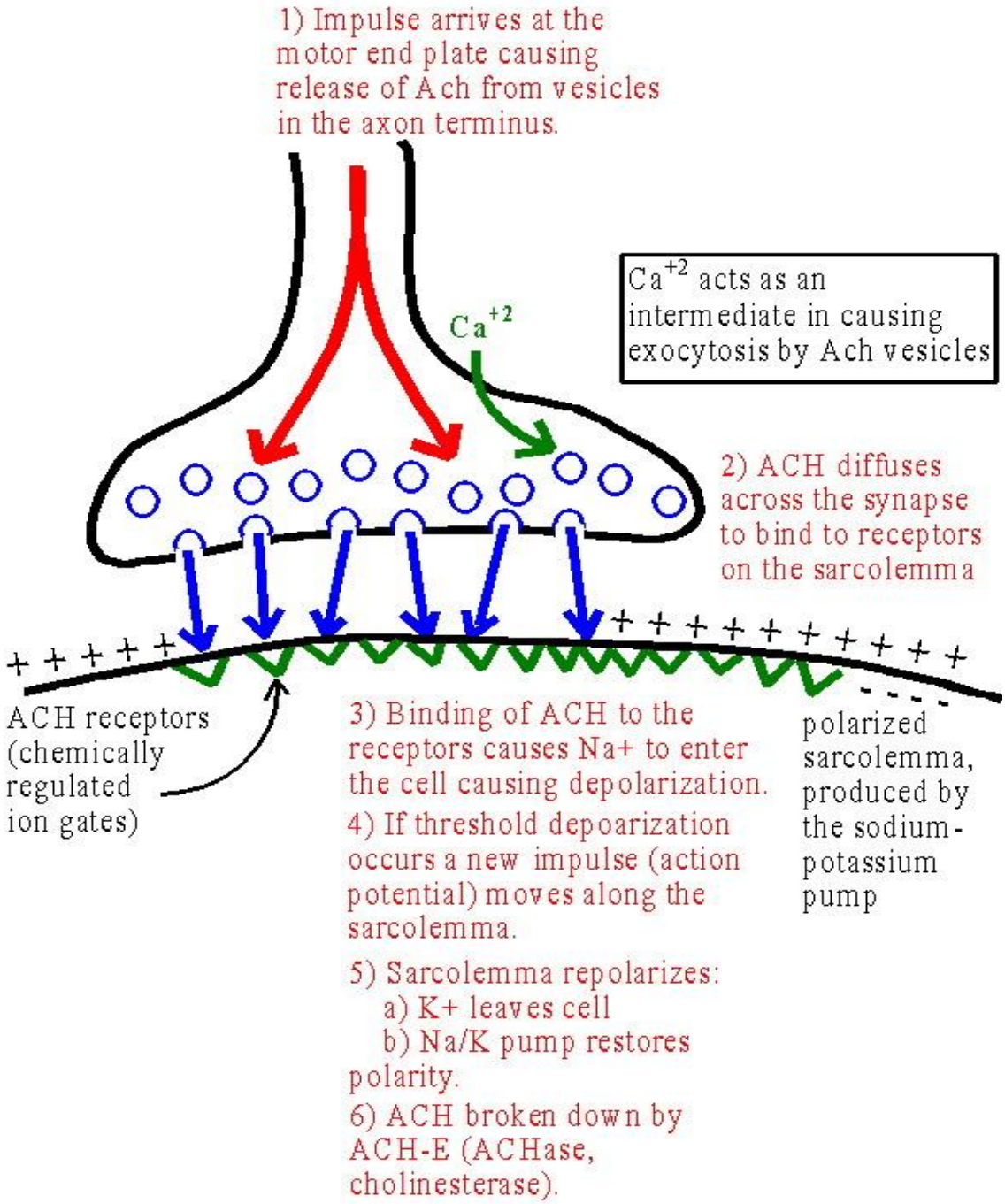
The acetylcholine receptor in junctional region is made of 5 subunits.it is attached to the membrane of the myocyte with the help of rapsin. the binding site of acetylcholine is present in α subunit. The α- 7 receptor contains 7 alpha subunits.

The acetylcholine receptor is a cylinder formed from arrangement of its subunit. Normally it is closed in position. once an agonist binds to the

both the α receptor it opens up leading to inflow of sodium, calcium and other neutral particles. Influx calcium leads to depolarisation and subsequent contraction of muscle.

If a molecule binds only one α site than that channel remains closed. It remains unavailable for action by agonist. This is the mechanism by which nondepolarising muscle relaxant works. It is a competitive blockade and thus a function of number of molecules available. Once cholinesterase inhibitors are given, the ratio of acetylcholine increases in the synaptic cleft and thus leads to recovery of muscle function.

Suxamethonium is made of two molecules of acetylcholine. Binding of suxamethomnium to neuromuscular junction causes contraction. As it is not destroyed immediately, the sodium channel that is open for a longer time becomes desensitized. This leads to relaxation of muscle.


1) Impulse arrives at the motor end plate causing release of Ach from vesicles in the axon terminus.
Ca+2 acts as an intermediate in causing exocytosis by Ach vesicles
Ca+2
2) ACH diffuses across the synapse to bind to receptors on the sarcolemma
ACH receptors (chemically regulated ion gates)
3) Binding of ACH to the receptors causes Na+ to enter the cell causing depolarization.
4) If threshold depoarization occurs a new impulse (action potential) moves along the sarcolemma.
5) Sarcolemma repolarizes:
a) K+ leaves cell
b) Na/K pump restores polarity.
6) ACH broken down by ACH-E (ACHase, cholinesterase).
polarized sarcolemma, produced by the sodium-potassium pump

NEUROMUSCULAR MONITORING

Neuromuscular junction can be monitored using a peripheral nerve monitor. Supramaximal stimulus is administered and various patterns are noted.

1. **Single muscle twitch**

   A single stimulus lasting just 0.2 millisecond in duration is given.

2. **Tetanic impulse**

   A prolonged stimulus of the frequency of fifty to hundred hertz lasting 5 milliseconds is given

3. **Train of four**

   Four stimuliat a frequency of two hertz each lasting 0.2 milliseconds.

4. **Double burst stimulation**

   3 short stimli lasting two hundred microseconds is delivered each separated by 0.02 seconds is given. This is followed by another two or three such stimuli at a interval of 0.75 seconds.

Characteristics of non-depolarizing block

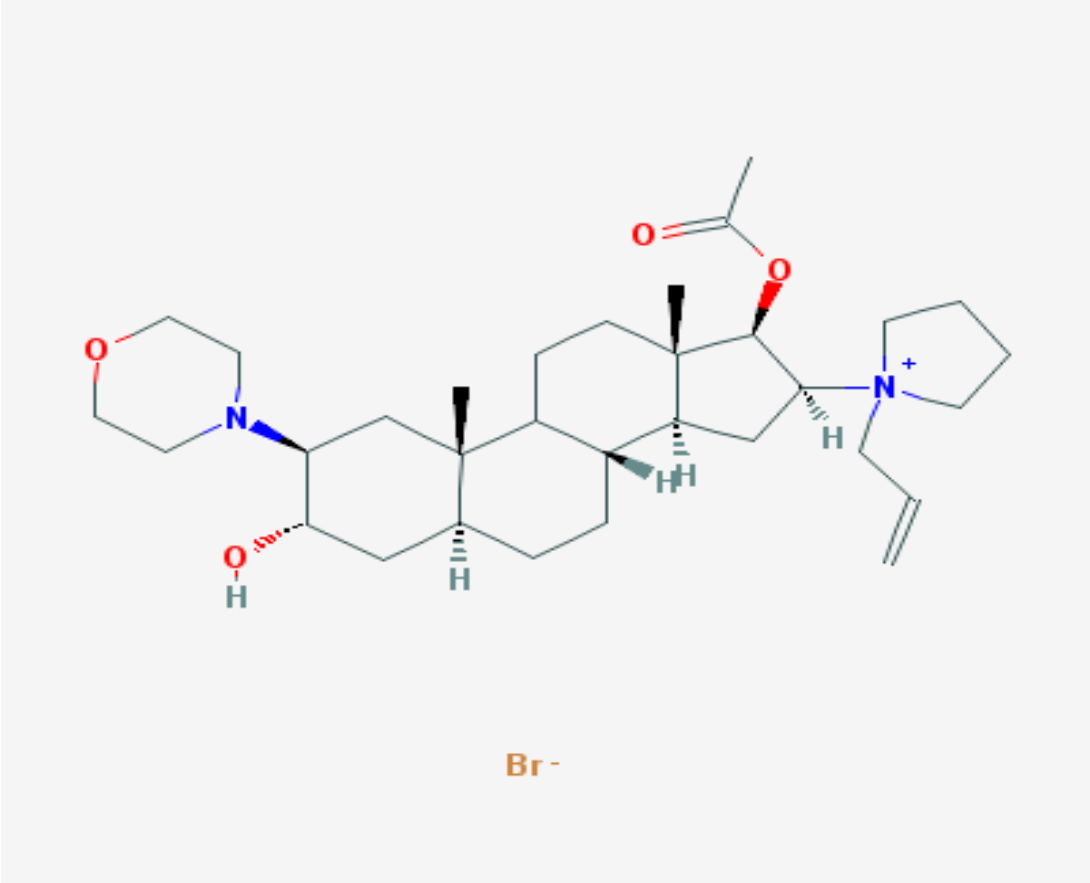
Non depolarizing neuromuscular blockade is characterized by fade. Fade is the diminution of evoked potential after prolonged or repeated stimulation of the nerve.

Absence of fade after repeated nerve stimulus correlates well with recovery. Double burst stimulation and post –tetanic indicate the recovery from the block, its better than single twitch and TOF counts.

PHARMACOLOGY OF ROCURONIUM

STRUCTURE:

It is a monoquartenary aminosteroid. Structurally it differs from vecuronium by the presence of hydroxyl group rather than an acetyl group in the A ring of steroid nucleus.



DOSAGE:

Its ED_{95} is 0.3 mg/kg, with onset of action around 2 minutes and duration of blockade of 20-30 minutes. When used in dosage of 3-4 times ED_{95} it can be used at par with suxamethonium for rapid sequence induction. When used at such high dosage it can cause blockade like a long acting nondepolarizing muscle relaxant with duration of action up to 60-90 minutes.

Large intramuscular doses -1-8mg/kg has been used in infants and children to permit rapid sequence induction at the cost of longer duration of blockade.

REASON FOR SHORTER ONSET OF ACTION:

It is a drug with low potency. When large number of molecules of such a low potency drug is administered, greater quantity of such a drug is available for diffusion and binding at the neuromuscular junction. Thus, a rapid onset of action is more likely to be achieved. So, Rocuronium when given in sufficient dosage can be used for suxamethonium, when the latter is contraindicated.

INTUBATING CONDITIONS:

Laryngeal adductor muscles and diaphragm are more resistant to neuromuscular blockade of non-depolarizing muscle relaxants[3] when compared to the adductor pollicis muscle. Hence, single twitch suppression at the level of adductor pollicis might not produce adequate intubating conditions.

When used at the dose of 0.6 mg/kg, Rocuronium does not produce good intubating conditions in 25% of patients.[1,5] Many authors have reported that IM Rocuronium given for RSI in infants and children does not consistently produce good intubating conditions.

OTHER PROPERTIES:

- It does not release histamine or known to cause any allergic reactions.
- It hasvagolytic properties and can be used in conditions such as opthalmoscopies and laparoscopies were vagal stimulation is present.

METABOLISM:

It is not actively metabolized and is excreted unchanged in bile. As it doesnot have any active metabolites, it can be better choice for infusions in ICU set up when compared to vecuronium.

Its duration of action is increased in liver failure and in pregnancy. Decreased hepatic clearance in elderly prolongs the duration of its action without affecting the time of its onset.

It should be used with caution in patients with renal failure.[3]Its duration of action is prolonged in morbidly obese patients when the dose of the drug is calculated using real bodyweight rather than ideal body weight.

USES:

Rocuronium is a unique drug among nondepolarizing neuromuscular blockers with rapid onset and hence is used in RSI.

DRUGS AFFECTING NEUROMUSCULAR BLOCKADE:

- VOLATILE ANAESTHETICS:

  - They produce dose dependent enhancement of blockade with nondepolarizing neuromuscular blockers
  - Enhancement of blockade is greatest with enflurane, isoflurane and sevoflurane and least with nitrous - opioid combinations.
  - It may be due to its central effect on CNS, which decreases muscle tone, or increased muscle blood flow, or decrease in sensitivity to depolarization in post junctional receptors.
  - Plasma concentrations required to produce suppression of twitch response is less in the presence of volatile anaesthetics.

- SYMPATHOMIMETIC DRUGS:

  - Drugs such as Ephedrine that increase sympathetic activity shorten the onset of neuromuscular blockers by increasing the cardiac output and muscle blood flow.

- On the contrary, drugs such as Esmolol by decreasing cardiac output and lengthens the onset time.

- ANTIBIOTICS

  - They may exert their effects by altering the prejunctional receptors thereby decreasing the release of acetylcholine.

- LOCAL ANAESTHETICS

  - At smaller doses, they enhance blockade of nondepolarizing neuromuscular blockers, but at higher concentrations have antagonistic effects.

- Diuretics and antiarrhythmic drugs can potentiate pre-existing blockade by decreasing the release of acetylcholine.

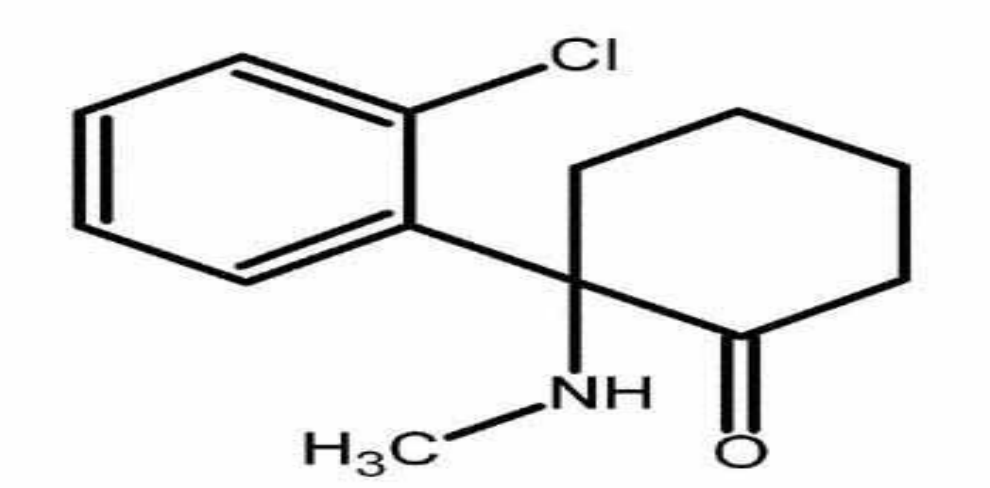
- Magnesium and lithium can inhibit the release of acetylcholine by binding to prejunctional receptors and potentiates the activity of muscle relaxants.

PHARMACOLOGY OF KETAMINE

Ketamine is a phencyclidine derivative which produces dissociative anaesthesia. The patient becomes cataleptic and non-communicative .It produces amnesia and intense analgesia. The preservative used is benzathonium chloride.

It is a drug with abuse potential; hence unauthorized use has to be avoided.

STRUCTURE ACTIVITY RELATIONSHIP



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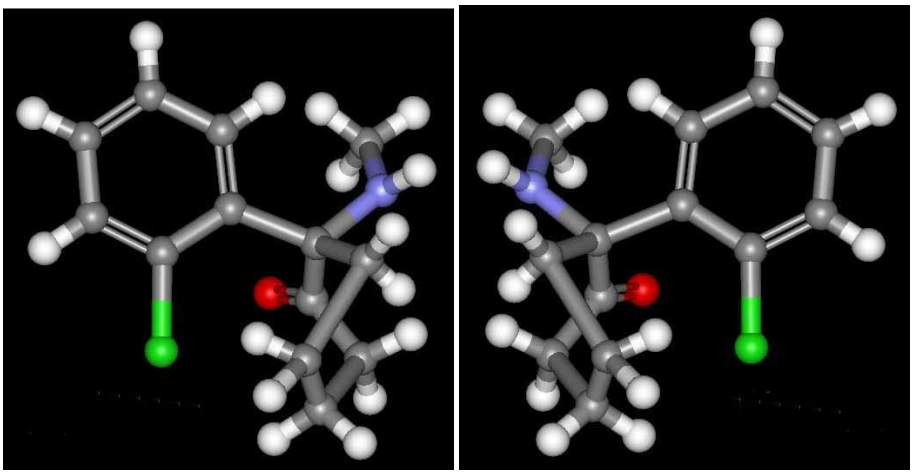
Ketamine is 2-(*o*-chlorophenyl)-2-(methylamino) cyclohexanone (hydrochloride):

Ketamine is water soluble. It has an asymmetric carbon atom leading to two optical isomers, S and R Ketamine.



S-(+)-Ketamine

R-(-)-Ketamine

S-Ketamine produces[1]

- more intense analgesia
- rapid metabolism and recovery
- less salivation and
- lower incidence of emergence reaction

MECHANISM OF ACTION

- Ketamine has an antagonist effect on NMDA receptors inhibiting the binding of glutamate,decreasingits presynaptic release and also potentiating the effect of GABA.

- Ketamine has agonistic action on kappa receptors and antagonistic effect on mu receptors.

- It may inhibit descending monoaminergic pathway thereby causing antinociceptive effect.

- Ketamine inhibits reuptake of catacholamines into postganglionic sympathetic nerve endings.

- It has antagonistic effect on muscarnic receptors.

- Has mild local anaesthetic like properties by its interaction with voltage gated sodium channel.

- Ketamine suppresses inflammatory response of neutrophils and improves blood supply .Cytokines inhibition may also contribute its analgesic effect.

MECHANISM OF ACTION –

CARDIOVASCULAR EFFECT

- Direct stimulation of CNS leads to increased sympathetic outflow thereby increasing cardiac output.
- Depressed baroreceptor reflex secondary to Ketamine leads to enhanced sympathetic outflow.
- Inhibition of reuptake of catecholamines.

PHARMACOKINETICS

It has rapid onset of action, short duration of action and high lipid solubility. Its pKa is 7.5. After administration, the Peak concentrations are attained after IV and IM injections at 1 minute and 5 minutes respectively.

It is not significantly bound to plasma proteins, crosses blood brain barrier, with as much as four to five times higher concentration seen in brain when compared to plasma. . It has high hepatic clearance (1 l/min), large volume of distribution (3 l/kg)and elimination half-life of 2 to 3 hours.

METABOLISM

Ketamine is demethylated by hepatic cytochrome p-450 into nor-Ketamine. Nor-Ketamine has one-fifth to one third of activity of Ketamine and can be responsible for prolonged effects of Ketamine especially when it is given in repeated doses or as infusions. Nor-Ketamine is then hydroxylated and conjugated and excreted by kidneys. Tolerance develops to Ketamine as it enhances its own metabolism.

CLINICAL USES

- **ANALGESIA:**
  - Subanaesthetic doses (0.2 mg/kg to 0.5 mg/kg IV) can produce intense analgesia. It is more for somatic pain than for visceral pain.
  - The site of action appears to be the thalamus and limbic system which interpret pain.
  - It can be useful adjuvant to opioids in low doses. Pain is also modulated by its action onopioid and NMDA receptors in spinal cord.

➢ INDUCTION OF ANAESTHESIA:

- IV dose is 1-2 mg/kg and IM dose is 4-8 mg/kg.
- Consciousness is usually lost in 30 to 60 seconds after IV and 2 to 4 minutes after IM administration.
- Normal airway reflexes are maintained even when patient becomes unconscious.
- Return of consciousness takes place within 10-20 minutes but proper orientation occurs only after 60-90 minutes.
- Amnesia persists for another 60-90 minutes. No retrograde amnesia is seen.
- IM induction is used in children and mentally retarded patients because of its rapid onset of action.
- Burns dressings, debridement, and skin grafting can be done using Ketamine as it has excellent analgesic properties and maintains airway which may be important in the presence of scar contractures and difficult airways.
- Tolerance may be noted on repeated usage.

- Induction in hypovolemic patients can be done because of its effects on sympathetic nervous system and its ability to

maintain blood pressure. Butit has to be used cautiously in a patient with heart disease as it can precipitate ischemia.

- Patients with bronchial asthma are benefited because of its bronchodilatory effects.
- It can be used in patients with malignant hyperthermia, but should be used with caution in patients with raised ICT, systemic or pulmonary hypertension and in acute intermittent porphyria.

➢ OTHER USES

- The neuraxial use is of limited value.5-50 mg in 3 ml saline intrathecally produces variable and brief analgesia.
- Ketamine is used to reverse tolerance to opioid analgesics due to its antagonism of NMDA receptors.
- It can be used in restless leg syndrome and mental depression.
- In subanaesthetic doses it can be used for bronchospasm.

SIDE EFFECTs

- CENTRAL NERVOUS SYSTEM:
    - Intracranial pressure is said to be raised in patients due to increase in cerebral blood flow by as much as 60% in the presence of normocapnia.
    - But recent studies suggest that Ketamine can be administered in anesthetised mechanically ventilated patients with mild elevation of ICT without adversely affecting the cerebral perfusion.
    - Thiopentone, benzodiazepines administered before Ketamine reduces the raise in ICT.
- CARDIOVASCULAR SYSTEM :
    - It resembles the stimulation of sympathetic nervous system. It causes increase in systemic and pulmonary blood pressure, increase in heart rate and myocardial oxygen demand.
    - There is increase in heart rate and systolic blood pressure for 3- 5 minutes after giving Ketamine which returns to basal levels only after 10-20 minutes. These hemodynamic alterations can be blunted with administration of benzodiazepenes or use of inhaledanaesthetics.

- VENTILATION AND AIRWAY :
    - Salivary and bronchial secretions can be increased if antisialogogue is not used along with Ketamine.
    - When used along with opioid medication it can cause respiratory depression.

- EMERGENCE DELIRIUM
    - Visual, auditory, tactile, proprioceptive and confusional illusion can occur with the administration of Ketamine during emergence from anaesthesia.
    - This can progress to frank delirium.
    - Transient cortical blindness may occur occasionally. Hallucinations and dreams can be there up to 24 hours. They have vivid colours and vivid content.

MECHANISM:

- Depression of inferior colliculus and medial geniculate nucleus leads to misinterpretation of auditory and visual stimulus.
- Loss of skin and musculoskeletal sensations, with decreased ability to perceive gravity, further increases this sensation.

INCIDENCE:

- Incidence is 5-30%.
- Predisposing factors are – age >15 years, female sex, dose > 2mg/kg, history of similar problems and use of atropine or droperidol as premedication.
- Its incidence decreases when it is repeatedly used.

PREVENTION:

- Benzodiazepines, inhaled anaesthetics and thiopentone decrease the incidence of delirium.

REVIEW OF LITERATURE

1.PelinTrajeTopcuoglu, MD et al[5] (Can J Anesth/J Can Anesth (2010) 57:113–119) investigated the effect of Ketamine and priming on the intubating conditions and the onset time of propofol- Rocuronium induction.

It was prospective, randomized double blind study with 120 ASA- I and II patients divided into 4 groups Ketamine, Ketamine-priming, priming and control. Ketamine at the dose of 0.5 mg/ kg or saline was given before induction or priming.

Priming was done with Rocuronium at the dose of 0.06 mg/kg 2 minutes before induction. This was done in priming group and Ketamine-priming group and saline was given in other two groups. No premedication was given as it was thought to influence the intubating conditions. Induction was done with propofol 2.5 mg/kg. Intubation was done after 1 minute.

They graded the intubating conditions according to the criteria established by Fuchs-Buder et al. The onset time of the neuromuscular blockade was noted at the disappearance of the last twitch in the TOF

monitor. They found that the time to reach TOF count of zero was significantly shorter with Ketamine (p =0.001) and not by priming (p=0.94).the onset time was216 ± 20 s inthe control group, 212 ± 27 s in the priming group,162 ± 18 s in the Ketamine group, and 168 ± 22 s in the Ketamine-priming group. The intubating conditions was improved with Ketamine (p=0.001) and not by priming (p =0.34).

Hence they concluded that small doses of Ketamine and not priming improved the intubating conditions and shortened the onset time of the neuromuscular blockade of Rocuronium.

2.Youngmi Kang, et al,[6]

studied effect of various dose of Ketamine on the onset time and intubating conditions of Rocuronium. 99 patients undergoing elective surgery were randomly allocated into 3 groups – control, K- 25, and K - 50.

It was a prospective randomized single blind study. Control group received 1 ml of normal saline while K 25 group received 0.25 mg/kg of Ketamine; K 50 received 0.50 mg/kg of Ketamine before induction. Intubation was attempted after one minute of induction, condition being noted as excellent, good or poor depending on the vocal cord position,

reaction to tracheal intubation and ease of laryngoscopy. The onset time of neuromuscular blockade was noted from time of administration of Rocuronium to zero twitch by TOF stimulation.

Onset time of Rocuronium in control group was 201 ± 103 sec, 136 ± 48 sec in the group K 25, 139 ± 36 sec in the group K 50. Ketamine shortened the onset with a P value of< 0.05.

There was no difference between groups K 25 and K 50.Intubating condition showed significant difference between the group C and group K 25 (P < 005) and group K 50 (P < 0.05). Again no significant difference was seen in between groups K 25 and K 50.

Hence they concluded that low dose Ketamine improved the intubating condition and the onset time of propofol Rocuronium anaesthesia. The dosing of Ketamine did not seem to affect the outcome.

3. Byung-RyangAhn et al[7] (Korean J Anesthesiol 2012 October 63(4): 308-313) investigated the effect of Ketamine and priming on the onset time of cis-atracurium in prospective double blind study.

120 patients undergoing general anaesthesia were divided into four groups – Ketamine, Ketamine-priming, priming and control. Patients in

priming group got cis atracurium at the dose of 0.01mg/kg IV and Ketamine group got Ketamine 0.5mg/kg IV.

The onset time was best for Ketamine-priming group (76.4 ±8 seconds) which was highly significant with p value of < 0.008. The onset was also accelerated in Ketamine and priming group when compared to control. The intubating conditions were excellent in Ketamine-priming group ($p<0.008$) when compared to other groups. It was better in Ketamine and priming group when compared to control.No difference was found between Ketamine and priming group.

Hence they concluded that Ketamine and priming can improve the intubating conditions. When used together they further improve these conditions and the onset time.

4 Ledowski T et al[8](Eur J Anaesthesiol. 2001 Aug;18(8):519-23),

They compared the effects of Fentanyl and s-Ketamine on the intubating conditions of Rocuronium. 90 patients who underwent elective surgery were studied. It was a prospective randomized double blind study. They were divided into three groups – control, Fentanyl (Fentanyl at the dose of 1.5 mcg/kg) and s-Ketamine(s-Ketamine the dose of 0.5 mg/kg).

They were induced with etomidate 0.3 mg/kg and Rocuronium 0.6 mg/kg. They were intubated at the end of 60 seconds and intubating conditions were noted as excellent, good or poor.

The intubating conditions in the Ketamine group was significantly better than control and Fentanyl group ($p < 0.001$).The heart rate and arterial blood pressure significantly rose in the Ketamine group compared to Fentanyl and control group ($p<0.001$).

The conclusion at the end of study was that s-Ketamine given before induction with etomidate and Rocuronium produced good intubating condition for RSI and can be used in the place of suxamethonium when it is contraindicated.

5.Leykin Y, et al.[9],.(Anaesth Intensive Care. 2005 Aug;33(4):462-8)

They studied the effect of priming and induction agent on the intubating conditions of Rocuronium. 60 ASA I-II patients undergoing elective surgery cases were studied. It was a prospective randomized study with four groups –control-Ketamine, priming-Ketamine, control-thiopentone, and priming-thiopentone.

Priming was done with 0.04mg/kg of Rocuronium 3 minutes before induction. Induction was with either thiopentone (4mg/kg) or Ketamine(1 mg/kg). Intubation was done 60 seconds after administration of Rocuronium. The intubating conditions were graded as excellent, good, fair or impossible to intubate. The onset of neuromuscular blockade was assessed with acceleromyography of the thumb.

The proportion of cases with good intubating conditions was significantly higher when Ketamine was used as induction agent ($p<0.05$). Further, the ratio of good to excellent conditions of intubation was better with the use of priming dose ($p<0.05$). There was no difference between the onset time and hemodynamic changes in these groups.

6.Anis S. Baraka, MD, et al[10](AnesthAnalg 1997;84:1104-7)

compared the effects of thiopentone and Ketamine on intubating conditions when used along with Rocuronium as induction agents in partiuents.

40 full term pregnant patients undergoing elective LSCS were included. It was a prospective randomized double blind study. They were divided into two groups of twenty each.

Premedication was done with 0.5 mg of atropine intramuscularly 30 minutes prior to induction. Opioids were given only after delivery of the baby. One group was induced with thiopentone 4 mg/kg and the other with Ketamine 1.5 mg/kg. Muscle relaxation was with Rocuronium at the dose of 0.6 mg /kg.

The time taken from the injection of Rocuronium to T1/control ratio of 50 % blockade, time taken for maximum blockade (onset time), and time taken for T1 to reach 25 % of control value (recovery time) were noted for each group. Tracheal intubation was attempted at 50 % neuromuscular blockade and the intubating conditions were graded according to Cooper's criteria. Time taken for 50% neuromuscular blockade, onset time, and recovery time and intubation condition were compared between the groups.

The time to reach 50 % blockade, the onset time (105 ± 35 s in the thiopentone group and 101 ± 35 sin the Ketamine group) and recovery

time were not statistically significant between the groups. However, the intubating conditions were excellent or good in all 20 cases in Ketamine group compared to 5 in thiopentone group and it was found to be significant.

The conclusion of the study was that KetamineRocuronium can be used for RSI in LSCS where suxamethonium is contraindicated.

7 .P. Hans, et al[11] (Anaesthesia, 1999, **54**, pages 266–296)

They compared the effects of thiopentone and Ketamine, as induction agents, on the intubating conditions while using Rocuronium for rapid sequence induction. 32 ASA I patients undergoing elective surgery were divided into two groups and effect of Ketamine and thiopentone was studied.

All patients received Alprazolam 1 mg orally one hour before surgery and 2mg midazolam was given 2 minutes before induction. Induction was done with thiopentone 5 mg/kg or Ketamine 2.5mg/kg followed by Rocuronium 0.6 mg/kg for muscle relaxation. Tracheal intubation was done after 60 seconds and intubating conditions were noted. The degree of neuromuscular block was assessed at the time of intubation as the percentage decrease in the height of T1 in TOF monitor.

The intubating conditions in Ketamine group was significantly better when compared to the thiopentone group (p=0.002). Vocal cord position and diaphragmatic movements were found to be better in patients in Ketamine group (p=0.002). The grade of neuromuscular blockade at the time of intubation did not differ much between the groups.

The study concluded that the intubation condition was better with Ketamine when compared to thiopentone.

7.WorawutLapisatepun, et al.[12](Chiang Mai Med J 2010;49(1):11-17.)

ina prospective double blind study compared the intubating conditionsbetween Ketamine and propofol induction. 80 ASA I –II patients undergoing elective surgery were studied.

Patients were divided into two groups to receive propofol 2.5mg/kg (group P) or Ketamine 2.5mg/kg (group K) followed by Rocuronium 0.45 mg/kg. Patients were not premedicated. After induction with agents as per their group, they were intubated at 75 seconds.

The intubating conditions were assessed using criteria devised by cooper et al., as excellent, good, fair or poor. The time taken for 100%

suppression of single muscle twitch was noted along with twitch height at 75 seconds. Hemodynamic changes were also noted for comparison.

All patients were intubated in the first attempt. The onset time of neuromuscular block was 71 seconds and 62.5 seconds in group K and P respectively, the difference was not statistically significant (p=0.46). The intubating conditions found to be acceptable was 90 % in group K and 85 % in group P, again not statistically significant (p=0.5). Hemodynamic comparisons showed that group K had significant rise in MAP (p=0.00) after induction, group P had significantly higher heart rate (p= 0.040) after induction.

Hence they concluded that Ketamine or propofol could be used for rapid sequence induction with good intubating conditions at 75 seconds after induction. Even though they expected Ketamine to produce better intubating conditions because of its better analgesic and sedative effects, they could not prove any statistical significance. The choice of the drugs was mainly for its hemodynamic effects.

9.Demet DOGAN EROL and CemilKAYA[13] **,**(EROL et al. Gynecol Obstetric 2012, 2:1,doi:10.4172/2161-0932.1000110)

They evaluated retrospectively the intubating conditions in 86 patients who received Rocuronium for muscle relaxation with propofol Ketamine induction. They concluded that even 0.4 mg/kg of Rocuronium with Ketamine at the dose of 1 mg/kg can produce good intubating conditions at 30 seconds.

10. Ji Young Kim et al[14](J AnesthDOI 10.1007/s00540-012-1485-4)

Studied the effect of alfentanil versus Ketamine on the hemodynamics and intubating conditions of Rocuronium bromide in children. 54 children aged between 3-9 years undergoing tonsillectomy were divided into 2 groups, alfentanil group (alfentanil 20 mcg/kg) and Ketamine group (Ketamine 0.5mg/kg). All patients were induced after one minute of giving the test drug with propofol 2.5 mg/kg and Rocuronium 0.3 mg/kg and anaesthesia was maintained with propofol. Intubation was done at 2 minutes.

The ratio of TOF compared to the basal reading was noted at 2 minutes. The parameters compared were neuromuscular blockade, intubating conditions and hemodynamic alterations.

The intubation condition and the twitch height at 2 minutes were not significantly different between the groups. The MAP arterial pressure was significantly higher after intubation in Ketamine group. They concluded that both Ketamine and alfentanil produce good intubating conditions with Rocuronium at the dose of 0.3 mg in children.

11 T. Ezri, .Szmuk R. D. et al[15] (Acta Anaesthesiologica Scandinavica Volume 47, Issue 9, pages 1067–1072, October 2003) studied the effect of alteration in cardiac output on the onset time of Rocuronium. They used noninvasive cardiac output monitor to find out the effect of Esmolol and Ephedrine.

The result was that the onset was faster with Ephedrine(52.2 ± 16.5 s) and longer after Esmolol (114.3 ± 11.1 s) as compared to Placebo (87.4 ± 7.3 s) ($P < 0.0001$).Cardiac output was significantly increased ($P < 0.05$) withEphedrine for 15 min after Rocuronium. With Esmolol, Cardiac Output decreased ($P < 0.05$) at 3 and 6 min.

Hence the conclusion was that by altering the cardiac output Ephedrine or Esmolol could affect the onset time of Rocuronium.

12.PeterSzmuk, MD, et al[16](AnesthAnalg 2000;90:1217–9)

They Hypothesized that Esmolol because of its decrease in cardiac output will prolong the onset of Rocuronium while Ephedrine will shorten its onset. The effect of Ephedrine and Esmolol on onset time of Rocuronium was studied.

The onset time of Rocuronium in placebo group was 93± 6 seconds,in Ephedrine group 66±46.6 seconds and in Esmolol group was 118.6± 11seconds.The onset time ofRocuronium was significantly shorter after Ephedrine (22%) and longer after Esmolol (26%), as compared toPlacebo.

They did not find any significant difference among the groups with respect to heart rate and hemodynamics. Hence they concluded that Esmolol prolongs the onset time of Rocuronium.

13 .D. W. Han, et al[17](Anaesthesia, 2008, 63, pages 856–860)

In their study, the effect of timing of administration of Ephedrine on the onset time of Rocuronium was noted. The administration of Rocuronium was donearound 4 minutes which coincides with the peak action of Ephedrineon the cardiac output.

They found out that onset of Rocuronium was shortened when given early (4 minutes before Rocuronium) by 20% (p=0.032) compared to late (30 seconds before Rocuronium) and control group. There was no difference in intubating conditions or duration of action.

They concluded that Ephedrine given 4 minutes before Rocuronium can be used in situations where suxamethonium is contraindicated and rapid intubation is required.

14.M. D. Gopalakrishna, et al[18]**{**British Journal of Anaesthesia 99 (2): 191–4 (2007) }

The effect of various doses of Ephedrine was compared with respect to the onset time of Rocuronium. They concluded that Ephedrine at the doses of 75 mcg /kg (p=0.003) to 100 mcg/kg (p=0.001) improved the

intubating conditions of Rocuronium without affecting the hemodynamics. Even though the onset time of neuromuscular blockade was faster with the administration of Ephedrine, it was not statistically significant.

15.Hernan R. Munoz, MD, et al[19] (AnesthAnalg 1997;85:437-40 437)

Effect of Ephedrine on the onset time of Rocuronium was studied. Ephedrine reduced the onset time to 72± 18seconds.When compared, it was to 92±31 seconds in placebo group(p=0.0006). Hence they concluded that Ephedrine reduces the onset time without adversely affecting the hemodynamics in healthy subjects.

16C. H. Tan, et al, [20]

concluded that Ephedrine in combination with propofol produced good intubating condition with Rocuronium than propofol alone in their study (Anaesthesia, 2002, 57, pages 223-226)**.**

17.YoungJu Won et al[21](Korean J Anesthesiol 2010 October 59(4): 244-248)

They Hypothesized that administration of phenylephrine will prolong the onset of action of Rocuronium. 64 patients undergoing elective surgery were divided into two groups- phenylephrine (0.9 mcg/kg) or saline.

Test drug was given five minutes before induction. The effect of the test drug on the onset of Rocuronium was compared. The onset time was delayed with phenylephrine. There was no difference between the groups with respect to heart rate, arterial blood pressure and intubating conditions.

18 .Anis Baraka MD, et al[22](CAN J ANAESTH 1992 / 39:2 / pp 135-8)

The effect of increase in cardiac output in pregnancy on onset time of neuromuscular blockade of vecuronium was studied. The onset time (that is, 50% neuromuscular blockade) was earlier in pregnant patients(80 ± 30 sec)than in the control group (144 ± 43 sec).the intubating conditions were adequate.

They concluded that the administration of vecuronium as per body weight produced rapid onset of neuromuscular blockade in pregnant woman.

19.GerardAudibert, MD et al[23](AnesthAnalg 1996;82:848-53)

In their study, they compared the onset time of mivacurium, vecuronium and Rocuroniumin an arm with reduced circulation caused by insufflations of a tourniquet.The onset time of maximal blockade of Rocuronium in the arm with tourniquet was half that of the perfused arm. This proved that the onset time of Rocuronium depended mainly on circulatory factors and not much on non-circulatory factors.

20Yong-guang Wanget al[23](J Pharm PharmaceutSci 10 (1): 53-60, 2007)

They studied the effect of hyperthyroidism on the onset time of Rocuronium.Eighteen hyperthyroid patients undergoing subtotal thyroidectomy were compared with eighteen euthyroid patients undergoing subtotal thyroidectomy.

They were induced with midazolam, Fentanyl and propofol. Rocuronium at the dose of 0.6 mg/kg was used for intubation and onset time compared. The onset time in hyperthyroid group was significantly shorter at 56± 10 seconds compared to 76 ± 13 seconds in euthyroid group($P<0.05$)

They concluded that the onset time was significantly shorter in hyperthyroid patient**.**

21 Z. Begecet al[24](Anaesthesia, 2009, 64, pages 282–286)

compared the effects of Ketamine and Fentanyl on the insertion conditions of proseal LMA. they Concluded that the hemodynamics was not altered with administration of 0.5 mg/kg of Ketaminebefore induction,. No difference in insertion conditions were noted between the groups.

22 Ittichaikulthol W et al [25] (J Med Assoc Thai. 2004 Mar;87(3):264-9).

In their study concluded that Ephedrine significantly shortened the onset time of Rocuronium ($p<0.001$) and can be used for Rapid Sequence Induction.

23 Santiveri X et al[26](Rev EspAnestesiolReanim. 2003 Apr;50(4):176-81)

concluded that premedication with Ephedrine reduces the onset time of Rocuronium and not that of atracurium.

MATERIALS AND METHODS

It was a randomized single blinded study conducted in Institute Of Anesthesiology And Critical Care, Madras Medical College, Chennai. After getting institutional ethical committee clearance and informed consent, 80 patients fulfilling the inclusion criteria were selected.

INCLUSION CRITERIA

- Age :18 – 60 years
- ASA : I & II
- Surgery : Elective surgery
- Who have given valid informed consent

EXCLUSION CRITERIA

- Not satisfying inclusion criteria.
- Patients with anticipated difficult intubation
- Increased risk of regurgitation
- History of cardiorespiratory illness
- Neuromuscular disorders
- Hepatic disease

- Renal disease
- known sensitivity to the drugs
- Pregnant females
- Emergency surgery

MATERIALS:

- Laryngoscopes of various sizes,bougie,oropharyngeal airway
- Drugs –

  propofol, Ketamine ,Rocuronium ,Fentanyl, glycopyrrolate, xylocard, normal saline, Ephedrine ,atropine, succinylcholine, midazolam,sevoflurane and other emergency drugs.
- Monitors –

  ECG, NIBP, SPO2, EtCO2, neuromuscular monitor (INMED nerve locator and peripheral nerve stimulator),
- 2 cc,5 cc and 10 cc syringe
- 18G intravenous cannula.
- Appropriate size endotracheal tubes
- Weighing machine

PARAMETERS NOTED WERE:

A. ONSET TIME

After induction, the ulnar nerve of the patient was stimulated and supramaximal stimulus was noted. The time taken between administration of Rocuronium and TOF count of zero was taken as onset time.

B. INTUBATION CONDITIONS

Intubation was done at 60 seconds and the intubation conditions were noted according to criteria established by cooper et al. The score is graded as excellent, good, fair or poor with respect to jaw relaxation, vocal cord position and diaphragmatic movements. If the vocal cord was adducted then the attempt was abandoned and another attempt made after 60 seconds.

GRADING OF INTUBAING CONDITIONS BY COOPER, ET AL

SCORE	JAW RELAXATION	VOCAL CORDS	RESPONSE TO INTUBATION
0	POOR	CLOSED	SEVERE COUGHING/ BUCKING
1	MINIMAL	CLOSING	MILD COUGHING
2	MODERATE	MOVING SLIGHTLY	DIAPHRAGMATIC MOVEMENT
3	GOOD	OPEN	NONE

\The Cormack lehane grading and Percentage Of Glottic opening

score were also noted.

<u>Cormack and lehanne grading was graded as :</u>

1. Visualization of entire vocal cords
2. Visualization of posterior part of laryngeal aperture

    2a - Visualization of posterior part of vocal cords

    2b - Visualization of arytenoids only

3. Visualization of epiglottis

    3a – epiglottis liftable

    3b – epiglottis adherent or only tip visible

4. No glottis structures seen

C. The hemodynamic alterations in the form of systolic blood pressure, diastolic blood pressure and mean arterial blood pressure were noted at various stages

- baseline,
- at the time of administration of premedication,
- at the time of administering test drug,
- after induction,
- after intubation,
- 1minute,3minutes and 5minutes after intubation

Heart rate variations during the same period were also noted for comparison.

CONDUCTION OF THE STUDY

After getting institutional ethical committee clearance, all patients posted for elective surgeries in general surgery and ENT theatres in our institute were chosen. Patients who had difficult airway and with other exclusion criteria were excluded. .After getting written informed consent, 80 patients of age group 18 to 60 years with ASA I and II, who satisfied our inclusion criteria ,were selected and randomly allocated into 2 groups using closed envelope method.

After connecting basic monitors- ECG,NIBP,SpO_2,temperature,baseline values were noted. Intravenous access was obtained with a 18 gauge venous cannula. All patients were given 10 ml/kg of normal saline before induction.

Inj Glycopyrollate 5 mcg/kg, inj midazolam 1 mg, inj Fentanyl 2mcg/kg and inj ondansetron 4 mg iv were given to all patients as premedication. All patients were preoxygenated with 100% Oxygen for 5 minutes.
Two minutes before induction, Group I received5 ml of normal saline, and groupII received Ketamine 0.5 mg/kg.

Induction was done using 2.5 mg/kg of propofol. As soon as the patient became unconscious, supramaximal stimulus was calibrated by stimulation of ulnar nerve and contraction of adductor pollicis muscle. TOF monitoring was done every 10 seconds.Inj Rocuronium at the dose of 0.6 mg/kg was administered.

Once the patient became apneic ,the patient was ventilated with 100% oxygen at 6 liters per minute and sevoflurane of 2%.Laryngoscopy was done at 60^{th} second of muscle relaxant administration. Patients were intubated at 60^{th} second with appropriate size oral endotracheal tube and intubating conditions were noted as per the criteria established by Cooper et al. if vocal cords were adducted or the intubating conditions were poor,

intubation was abandoned. Patients were ventilated with 100 % oxygen and 2% sevoflurane for another minute .Reintubation was tried 60 seconds later.

The heart rate,blood pressure and mean arterial pressure were recorded at the time of administering premedication and administration of test drug. Same parameters were noted after induction, after intubation, thereafter 1,3 and 5 minutes following intubation. Cormack lehanne grading and percentage of glottis opening score was also noted.

Intubating conditions was assessed as excellent, good, and poor using criteria established by cooper et el.

The data collected was analyzed using statistical package for social sciences (SPSS version 15). All qualitative variables were compared using chi square test and quantitative variables using students't' test.

It was calculated that 34 patients per group were required for a 0.05 level of significance and 80% power to detect at least a 50% difference between the control group and the other group with respect to acceptable intubating conditions.

we chose a study with 40 patients in each group.

OBSERVATIONS AND RESULTS

This was a prospective, randomized, single blinded study done to assess the effect of Ketamine on the onset time of Rocuronium.

The primary end point was the onset of neuromuscular blockade and the quality of intubation. Secondary measures done were the heart rate and hemodynamic changes that occurred during the course of intubation

Statistical analysis was done using statistical package for social sciences (SPSS for windows, version 15). Results are represented here as mean and standard deviation.

All qualitative variants were compared using Chi Square test. Quantitative variants using students‘t’ test. A ‘p’ value of less than 0.05 was considered significant.

80 patients were included in this study. Group A is SALINE group and Group B is Ketamine group.We were able to intubate all the patients in the first attempt. There demographic characters were all comparable between groups.

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COMPARISON OF DEMOGRAPHIC PROFILE

Table 1. Demographic profile: AGE(IN YEARS)

Group	Number	Mean	SD	P value
A	40	32.38	11.302	0.866 N.S
B	40	31.95	11.161	

The mean age of patients in control group was 32.38 ±11.32 years and in Ketamine group was 31.95 ±11.16 years. Hence they are comparable with respect to age**.**

Table 2. Demographic profile: SEX:

Group	Male		Female		P value
	No	%	No	%	
A	17	42.5	23	57.5	820 **N.S**
B	16	40.0	24	60.0	

With regard to sex distribution between the groups, both were comparable and there was no statistical difference between the two groups (p=0.820)

COMPARISON OF BMI

In our study we compared the weight and BMI of the patients in the two groups,as they can influence the intubating conditions. The height, weight and BMI were comparable between the groups and there was no statistically significant difference between the two groups.

Table 3. DEMOGRAPHIC PROFILE: WEIGHT (IN KG)

Group	No	Mean	SD	P value
A	40	57.23	9.986	.216 N.S
B	40	54.80	7.183	

Table 4: Demographic profile: height (IN cm)

Group	No	Mean	SD	P value
A	40	160.28	9.378	.094 N.S
B	40	159.88	8.275	

Table 5. Demographic profile: BMI:(KG/ m^2)

Group	No	Mean	SD	P value
A	40	21.391	1.8010	.080 N.S
B	40	22.110	1.8190	

COMPARISON OF EASE OF INTUBATION

Hemodynamic alterations of intubation not only depend on the degree of relaxation of the vocal cords but also on the anatomic variations that make the intubations easy or difficult. Thus we measured modified Mallampatti score,Cormack lehane grading and the Percentage OfGlottic opening as a measure of difficulty of intubation.

Table 6. Distribution of MMS grade:

Group	MMS I		MMS II		MMS III		P value
	No	%	No	%	No	%	
A	19	47.5	19	47.5	2	5	.280 N.S
B	13	32.5	26	65	1	2.5	

MMS score was not significantly different between the two groups. The p value is 0.280.

Table 7. Distribution of POGO score:

Group	mean	S D	P value
A	81.93	26.383	0.258 N.S
B	87.88	19.855	

The Cormack lehane grading and POGO score did not differ significantly between the two groups.

Table 8. Distribution of CL grade:

Group	I		II A		II B		P value
	No	**%**	**No**	**%**	No	%	
A	25	62.5	11	27.5	4	10	922 N.S
B	26	65	11	22.5	3	2.5	

COMPARISON OF INTUBATING CONDITIONS

Patients were intubated at 60 seconds and intubation conditions noted as per cooper's criteria. We were able to intubate all the patients orally at 60 seconds using appropriate size McIntosh blade and endotracheal tubes.

Our finding were

- Excellent intubating conditions were seen in 22 patients in saline group and 25 patients in Ketamine group,
- good intubating conditions were seen in 10 patients in saline group and 10 in Ketamine group,
- While fair intubating conditions were seen in 8 patients and 5 patients respectively.
- Both the groups did not have any poor intubating condition

Table 9. Distribution of intubating conditions:

Grade	Group A		Group B	
	Number	percentage	number	percentage
excellent	22	55	25	62.5
Good	10	25	10	25
Fair	8	20	5	12.5

Table 10 :Acceptable intubating conditions

Group	**excellent**	**Good**	**Acceptable Conditions**		**P value**
	No	**No**	No	%	
A	22	10	32	80	0.642 N.S
B	25	10	35	87.5	

Excellent and good intubating conditions were taken acceptable. In Ketamine group it was 87.5% and in saline group it was 80%.Statistical analysis done of intubating conditions using chi-square test showed no statistically significant difference between the groups. The ‘p’ value was 0.642.

Table 11. time required for onset of blockade: (IN SECONDS)

Group	No	Mean	SD	P value
A	40	127.5	48.758	**.001 Significant**
B	40	93.48	33.749	

The mean onset time of neuromuscular blockade was statistically significant in Ketamine group than with saline group. The mean duration with Ketamine group was 93.48± 33.74 seconds versus 127.50±48.75 seconds in saline group. The 'p' value is 0.001 and it is statistically significant.

COMPARISON OF HEMODYNAMICS

The hemodynamic alterations were noted and compare between the groups. There was no statistically significant difference in the systolic and diastolic blood pressure between the groups throughout the study period.

Table 12 : comparison of mean of systolic blood pressue (mm Hg)

	Group A		Group B		P value
	Mean	S D	Mean	SD	
Basal	123.30	15.061	119.03	13.766	0.189
Premed	122.25	13.272	115.95	11.489	0.26
After test drug	121.95	13.142	120.28	12.677	0.563
After induction	108.43	16.295	112.25	14.961	0.278
After intubation	135.03	16.482	129.85	17.222	0.174
1 minute	126.65	18.524	125.03	16.247	0.678
3 minutes	120.18	14.970	118.98	16.285	0.732
5 minutes	120.13	13.758	115.93	15.669	0.207

Table 13 : comparison of mean of diastolic blood pressue (mm Hg)

	Group A		Group B		P value
	Mean	S D	Mean	SD	
Basal	77.55	8.327	74.65	6.475	0.189
Premed	76.38	6.736	73.28	6.801	0.26
After test drug	76.85	7.113	74.58	5.134	125.03
After induction	70.25	11.038	71.72	9.902	0.278
After intubation	85.93	14.995	85.13	12.999	0.174
1 minute	82.55	10.085	80.70	16.128	0.678
3 minutes	77.22	9.112	74.80	10.821	0.732
5 minutes	75.35	10.187	75.38	10.280	0.207

COMPARISON OF MAP OF 80 PATIENTS

But, comparison of mean arterial blood pressures of the two groups showed that MAP was well maintained in the Ketamine group

Table 14. COMPARISON OF MAP OF 80 PATIENTS (mm Hg)

	Group 1		Group 2		P value
	Mean	SD	mean	SD	
Basal	92.80	8.43	89.44	7.493	0.064
Premeded	91.67	7.62	87.50	6.688	0.011
Test Drug	91.88	7.53	89.80	6.120	0.181
Induction	82.98	11.87	85.23	10.69	0.374
Intubation	102.29	13.98	100.03	13.42	0.463
1 min	97.25	15.76	95.48	10.80	0.559
3 min	91.54	10.89	89.53	10.45	0.401
5 min	90.27	10.05	88.89	10.97	0.559

The fall in mean arterial pressure in the Ketamine groupafter induction when compared to baseline was 4.2 mm hg. The fall in mean arterial pressure in saline group was 9.82 mm Hg. This was statistically significant at P=0.036.

The mean arterial pressures at 5 minutes after intubation in both the groups were comparable. The change in mean arterial pressure from baseline at 5 minutes in both the groups did not differ significantly (p= 0.444)

Table 15 Comparison of change in MAP from baseline MAP (mm Hg)

	Group I	Group II	P value
Induction	- 9.82	- 4.20	0.036
5 minutes	- 2.52	- 0.55	0.444

HEART RATE VARIATIONS

Heart rate was noted between the two groups throughout the study and compared .it was comparable between the groups in this period at all times.

Table 16. COMPARISON OF HEART RATE OF 80 PATIENTS (mm Hg)

Group	No	BASELINE		INDUCTION		5 MIN	
		Mean	SD	Mean	SD	MEAN	SD
A	40	82.43	10.49	87.53	14.05	90.27	10.05
B	40	84.98	8.65	91.05	11.99	86.50	11.42

DISCUSSION

In our study, we found that addition of Ketamine at the dose of 0.5 mg/kg given before induction shortened the onset of Rocuronium 0.6 mg/kg. Even though the intubating condition was acceptable and the percentage of excellent intubating conditions was around 62.5 % with Ketamine, we could not find any statistical difference with the control group.

Rapid sequence induction requires use of agents which have shortest onset of action thereby limiting hypoxia and chance of aspiration. Suxamethonium has traditionally been used for rapid sequence induction. When suxamethonium is contraindicated, Rocuronium is the next choice. But at doses of 0.9-1.2 mg/dl , the duration of action is prolonged.

To overcome this various strategies have been used to reduce the onset time and improve the intubating conditions while using a lower dose of 0.6 mg/dl. One such method is to maintain the cardiac output during induction. **Gerard Audibert, MD, et al**[23] concluded in their study that for low potency drugs like Rocuronium the onset depends more on maintaining circulation to the muscle and not on local factors.

Study conducted by **Yong-guangWangetal**[29] in hyperthyroid patients in whom cardiac output is increased also gives credence to the use of this strategy. They found that onset time in hyperthyroid patients is shorter when compared to that of euthyroid patients.

In our study, we used Ketamine at a low dose of 0.5 mg/kg to increase the sympathetic outflow. This will blunt the fall of blood pressure caused by propofol and maintain the cardiac output. Also ,by raising the catecholamine levels, it will enhance the blood flow to muscleby increasing the $beta_2$ activity.Hence its action is twofold.

In our study, we found that the onset time was significantly shortened with the use of Ketamine. The onset time in Ketamine group was 93.48± 33.74 seconds versus 127.50±48.75 seconds in saline group. The 'p' value is 0.001.

These findings were similar to that of **PelinTrajeTopcuoglu, et al.**[5]the onset time was significantly shorter with Ketamine(p =0.001) not by priming (p=0.94).216 ± 20 s inthe control group, 212 ± 27 s in the priming group,162 ± 18 s in the Ketamine group, and 168 ± 22 s in the Ketamine-priming group.

We found that the onset time was shorter in both groups than their study. The reason could be that we used Fentanyl and sevoflurane at the time of induction. Several studies suggest that sevoflurane can shorten the onset of Rocuronium.[30,32]

Youngmi Kang, M.A.,etal[6]findings also concur with ours. They did not use any opioids or volatile anaesthetics before intubation.Onset time of Rocuronium in control group was 201 ± 103 sec, 136 ± 48 sec in the group K 25, 139 ± 36 sec in the group K 50.the onset time was significantly shortened with p value of < 0.05.

Byung-RyangAhnet al[7]compared Ketamine and priming on the onset time of cis-atracurium. Their study also correlates with our study. The onset time was best with Ketamine – priming group (76.4 ±8 seconds) which was highly significant with p value of < 0.008.Ketamine or priming alone also reduced the onset time of cis-atracurium when compared to control group.

Leykin Y, et al.,[9]could not demonstrate any difference between the thiopentone group and the Ketamine group with regard to the onset time**.** They used a total dose of 0.4 mg/kg of Rocuronium, which is around one ED_{95}. Since the onset depends upon the number of molecules at neuromuscular junction especially for low potency drugs like

Rocuronium.[1,2] The use of twice ED_{95} (0.6 mg/kg) might have decreased the onset time.

Anis S. Barakaet al[10] also differed from our study. They compared the effects of induction agents in pregnant ladies on the onset time of Rocuronium. They could not find any significance between the thiopentone and Ketamine groups. The time taken from the injection of Rocuronium to T1/control ratio of 50 % blockade and time taken for maximum blockade (onset time) was not different. Since pregnant patients already have increased cardiac output as their altered physiology it is possible that addition of Ketamine did not alter the onset time.[22]

Similar to Ketamine, Ephedrine an indirect acting sympathomimetic will increase the cardiac output and is expected to reduce the onset time. Esmolol a beta blocker similarly due to its effect on cardiac output will prolong its onset time.

T. Ezri, .Szmuk*, R. D. et al[15]compared the effect of Esmolol and Ephedrine on the onset time of Rocuronium, they found that Ephedrine reduced the onset time ($P < 0.0001$), and Esmolol prolonged the onset time ($P < 0.05$). They monitored the cardiac output and found it to be significantly increased in Ephedrine and decreased with Esmolol. This corresponded with their clinical finding.

C. H. Tan, et al,[20]Hernan R. Munoz, et al,[19]Gopalakrishna, et al,[18]D. W. Han, et al,[17] and Peter Szmuk, et al[16] also published similar results.

Young Ju Wonet al[21]studied the effect of phenylephrine on the onset time of Rocuronium. Phenylephrine though maintains the mean arterial pressure by increasing the peripheral vascular resistance, it does not alter the cardiac output or the blood supply to the muscles. Thus it is not expected to shorten the onset. In fact, in their study the onset time was delayed compared to saline.

Ketamine has anesthetic properties.[1,2,5] By providing analgesia and hypnosis, airway reflexes are obtunded and hence, it can improve the intubating conditions. Thus we hypothesized that Ketamine not only shortens the onset but also provides better intubating conditions.

In our study, we were able to intubate all the patients in first attempt. Acceptable intubation conditions were found in 87.5 % of patients in Ketamine group versus 80 % of patients in saline group. We could not demonstrate a significant difference between the two groups, (p=0.641)..

WorawutLapisatepun, et al[12]results were similar to ours**.** The percentage of acceptable intubating conditions was 90% in Ketamine group compared to 85% acceptable intubating conditions in propofol group. They could not demonstrate a significant difference between the two groups (P =0.499)

Ji Young Kim et al[14]study also concurs with our study. They compared low dose Ketamine and alfentanil on the intubating conditions of Rocuronium. The percentage of acceptable intubating conditions and the mean twitch height at the time of intubation was similar between the two groups and no significant difference could be found between the groups (p=0.326).

Z. Begecet al[24]compared the effect of alfentanil and low dose Ketamine on insertion conditions for proseal LMA. They could not demonstrate any statistical difference between the insertion conditions.

Results of the study done by**PelinTrajeTopcuoglu, et al**[5]differ from our study. The intubating conditions were acceptable in 96.6% of cases in Ketamine group and 90% of cases in Ketamine –priming group , which is similar to our findings.

But in the control group, the percentage of acceptable intubating conditions was 53.3% and in the priming group it was 66.6%.Hence they

concluded that intubating conditions were better with Ketamine and not priming. This differs from our findings.The reason could be that we used Fentanyl as premedication and sevoflurane before intubation in our study which could have affected both the groups in providing better intubating conditions.

Byung-RyangAhnetal[7]also used low dose Ketamine and priming to improve the intubating conditions of cis-atracurium .they concluded that both Ketamine and priming improved the intubating conditions.

Leykin Y, et al[9]study concluded that Ketamine improved the intubating conditions when compared to Fentanyl or priming. Their result differed from that of our study. The reason for such a difference could be that they used a higher dose of Ketamine (1mg/kg).

Hemodynamic alterations between the groups were also compared in our study. The rapid distribution of the drug to the neuromuscular junction was due to the maintenance of hemodynamics of Ketamine.

In our study, there was fall in mean arterial pressure after induction in both the groups. The fall was less (-3.12 mm Hg) and mean arterial pressure was better maintained in Ketamine group compared to saline group (-9.82 mmHg) with a p value of 0.036.

The heart rate were also comparable throughout the study period and did not achieve level of statistical significance

PelinTrajeTopcuogluetal[5]concur with our study.The decrease in mean arterial blood pressure in Ketamine group was significantly less in Ketamine groups when compared to others (p=0.001).

The heart rate was comparable between the Ketamine and non-Ketamine groups without any statistical significance (p=0.095) .

Even though the mean blood pressures and heart rates were higher in Ketamine groups compared to non-Ketamine groups, **Byung-RyangAhnet al**[7]could not demonstrate any significant difference between these two groups.

Ledowski T et al[8]found that the heart rate and arterial blood pressures rose significantly in Ketamine group when compared to Fentanyl (p<0.001)

Leykin Y, et al[9]did not find any significant differences in hemodynamics between the Ketamine and thiopentone groups**.**

P. Hans et al[11]in their study, report that there was 11% raise in systolic blood pressure (p=0.0006) and 10% mean aterial pressure in Ketamine

group when compared to thiopentone group just before intubation when compared to the to the baseline values.

Such a raise in blood pressure could be due to the large dose of Ketamine (2.5 mg/kg) used in this study .the heart rate changes were not significant between the groups.

Worawut Lapisatepun, et al[12]also reported similar findings. As they also used higher dose of Ketamine (2.5 mg/kg).Hemodynamic comparisons showed that group K had significant raise in MAP (p=0.00) after induction, while group P had significantly higher heart rate (p= 0.040).

SUMMARY:

From this prospective single blind study conducted on 80 patients undergoing elective surgery, we tried to evaluate the effect of the Ketamine on the onset time and intubating conditions of Rocuronium bromide. The summary of our findings are

1) The demographic profile such as age , sex ,height ,weight and BMI were comparable between the groups.
2) The indices that measure the difficulty of an intubation such as MMS, Cormack Lehane grading and POGO score were comparable between the groups.
3) The mean time of onset of neuromuscular blockade was 93.48± 33.74 seconds in Ketamine group versus 127.50±48.75 seconds in saline group. It was statistically significant with a ‘p’value of0.001.
4) The percentage of acceptable intubating conditions in Ketamine group was 87.5% and in saline group was 80 %. Both the groups were comparable.
5) When compared to the baseline values, the mean arterial pressure at induction was better maintained in Ketamine group than in saline group.
6) Arterial blood pressure and the heart rate at other times were comparable between the groups.

CONCLUSION:

From our study we conclude that administration of low dose Ketamine before induction reduces the onset time of Rocuronium bromide with stable hemodynamics. Though, acceptable intubating conditions were seen in 87.5 % of cases with Ketamine, we could not demonstrate a significant difference between the groups.

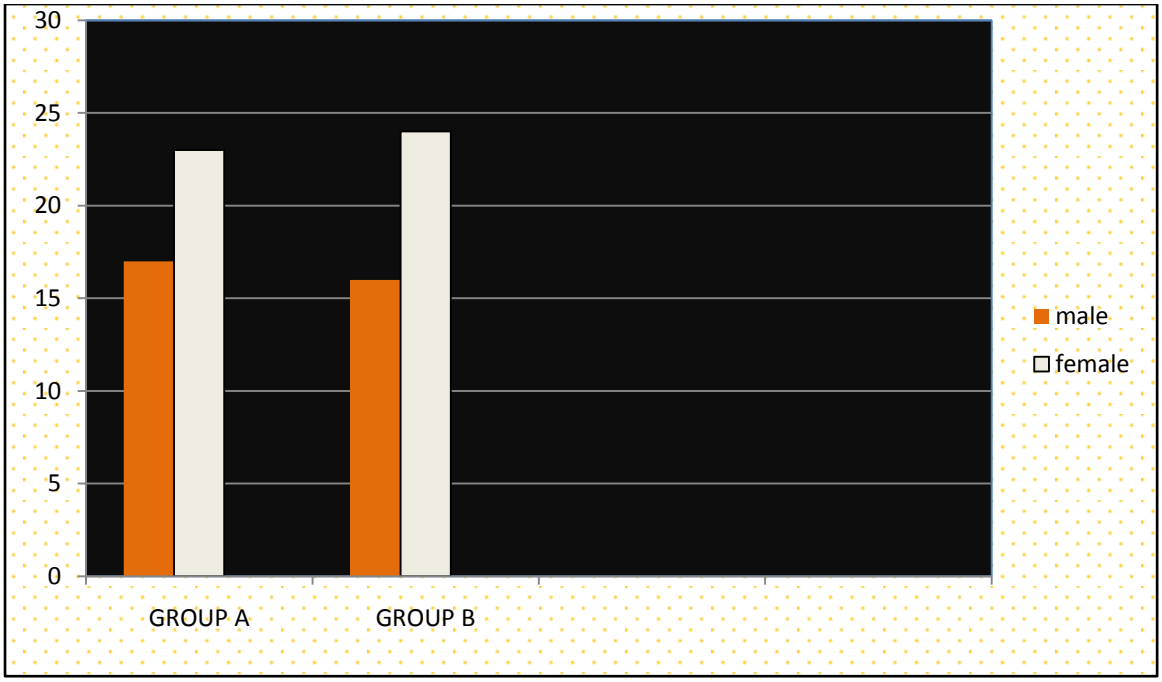

Fig 1 : sex distribution between the groups

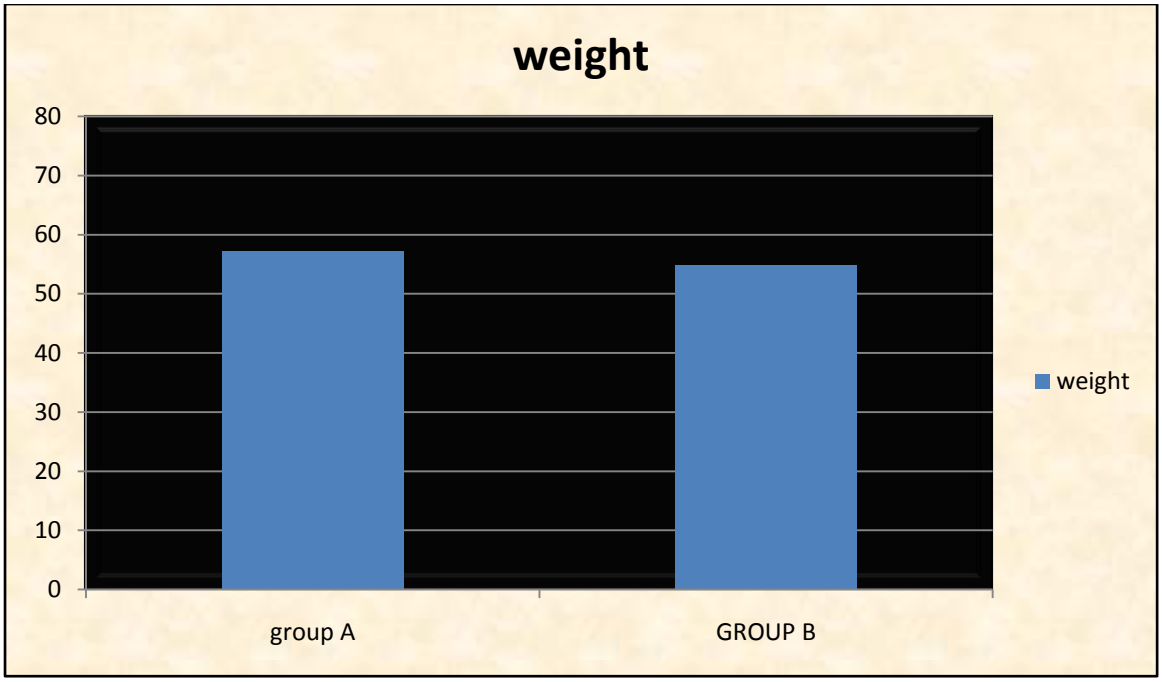

Fig 2 : weight distribution between the groups

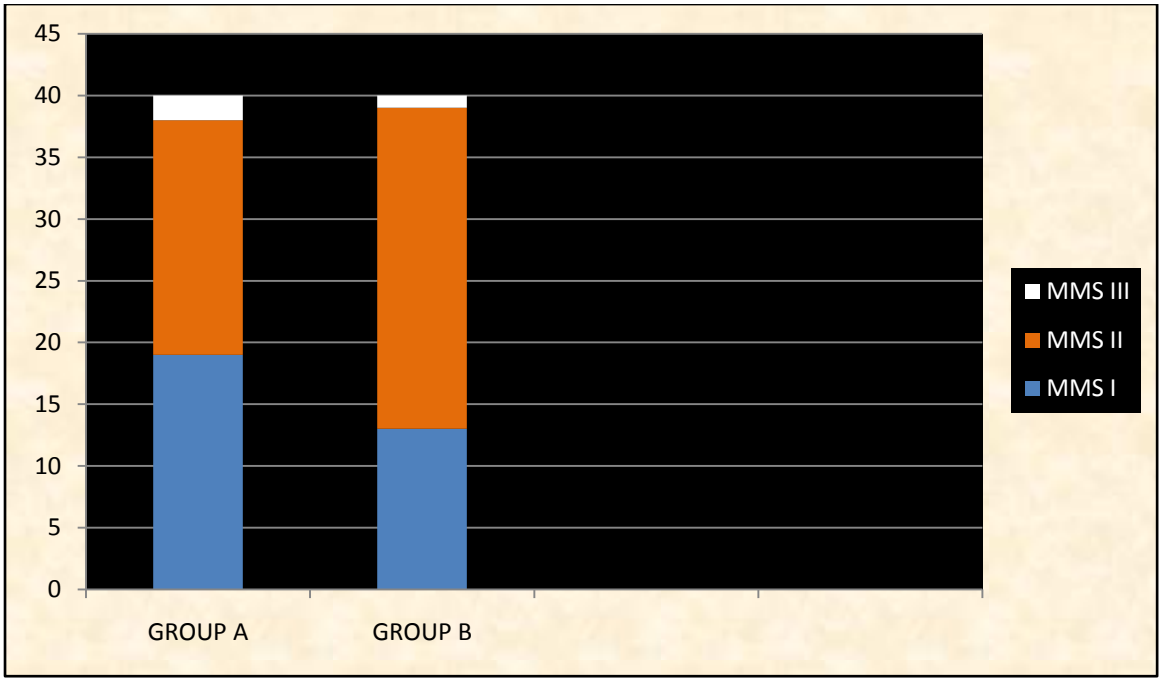

Fig 3: distribution of MMS classes

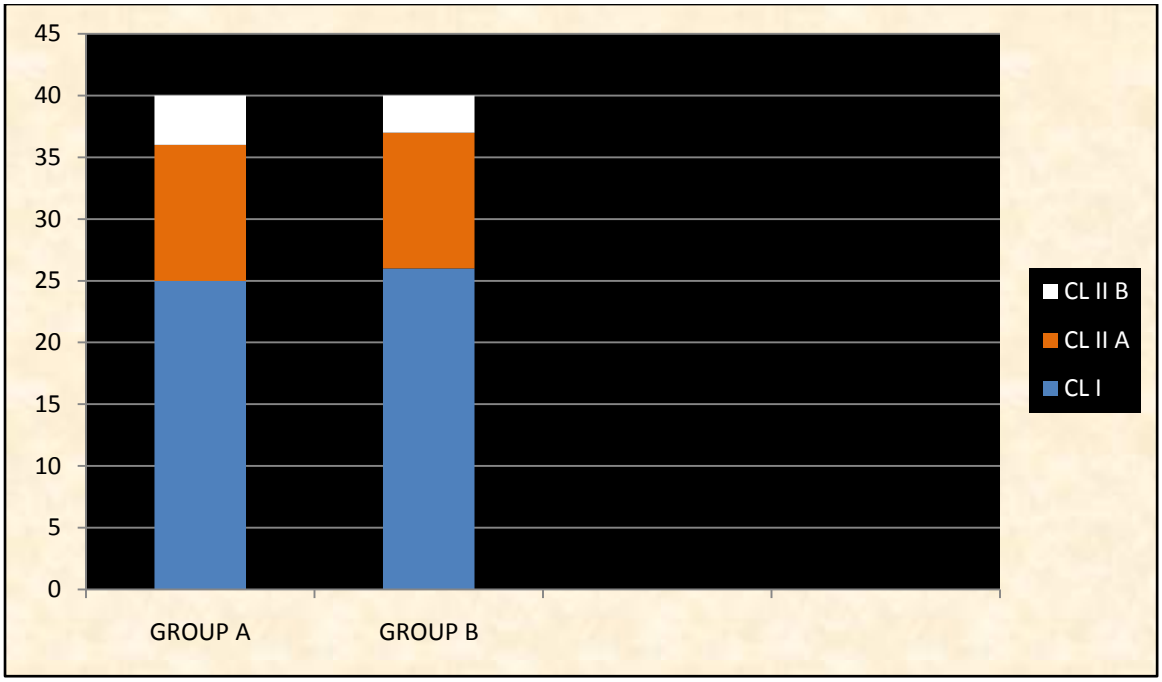

Fig 3: distribution of CL grades

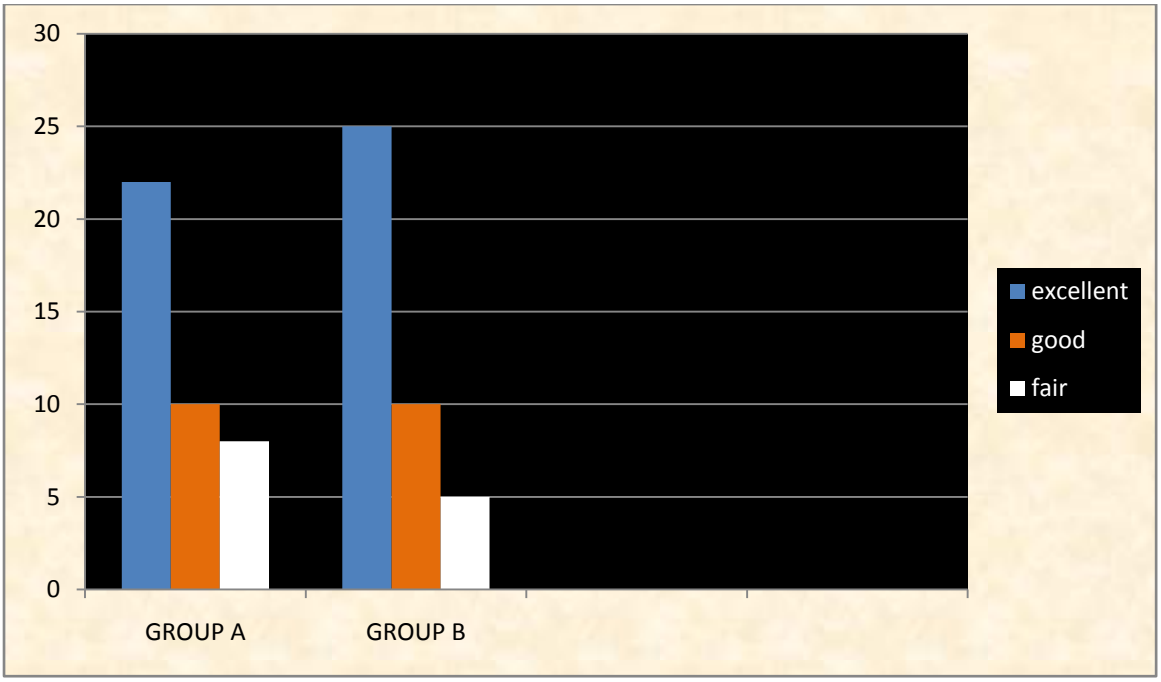

Fig 5: comparison of intubating conditions

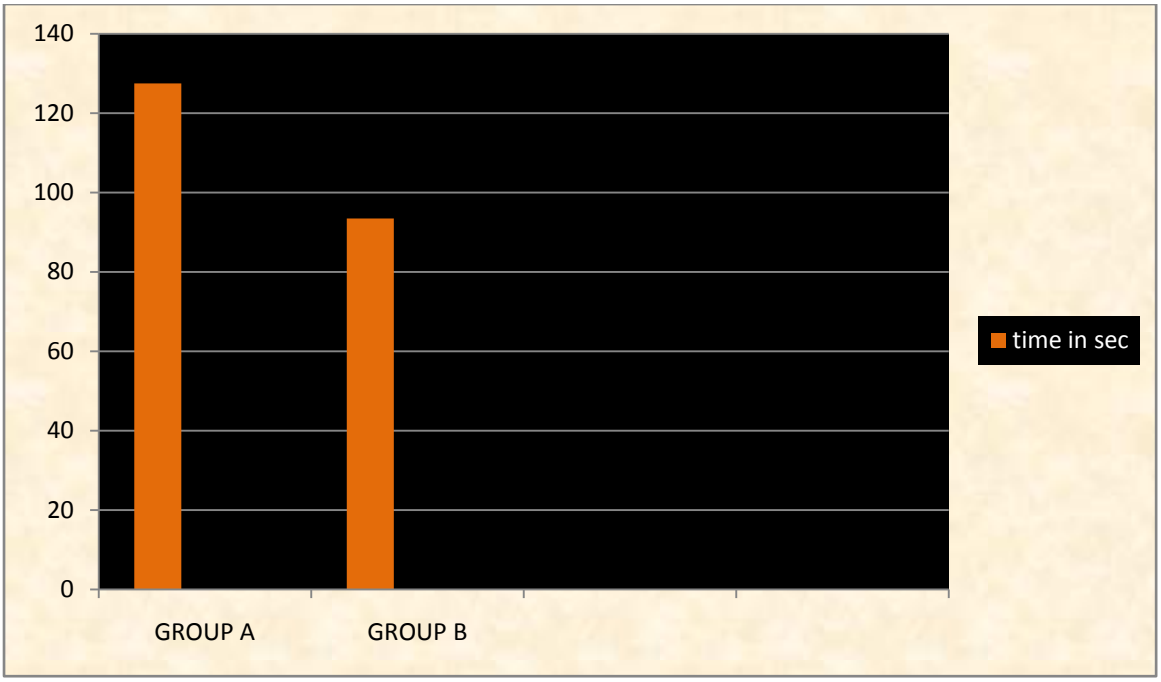

Fig 6 : onset time of neuromuscular blockade

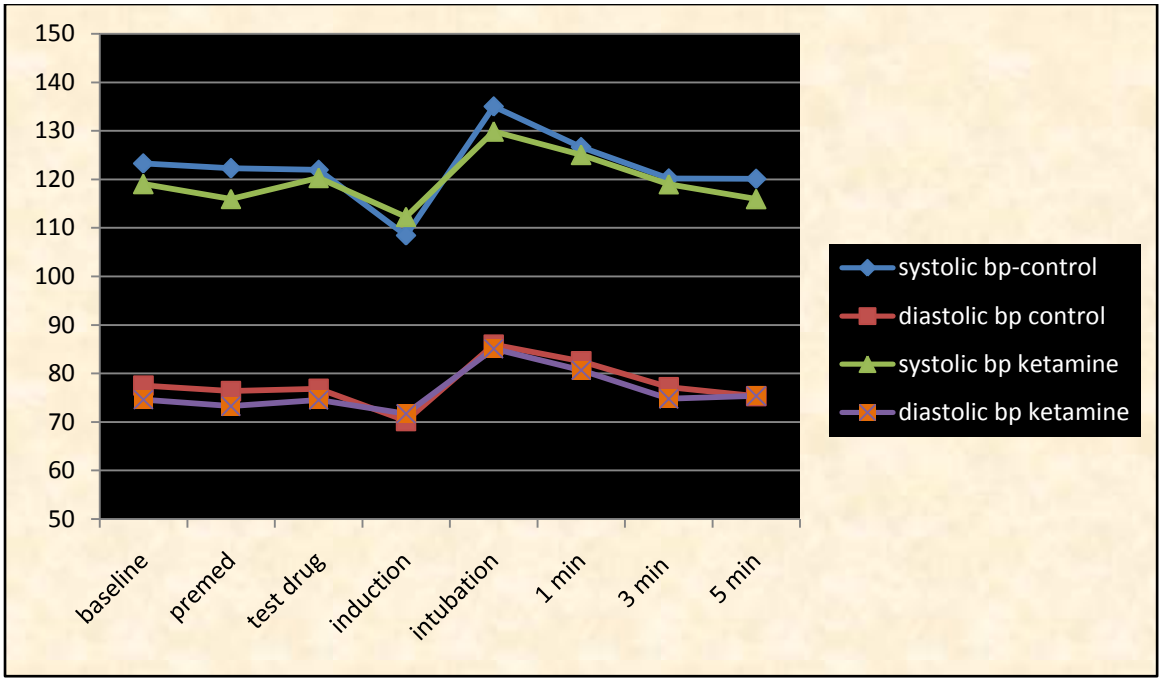

Fig 7: line diagram showing the variations in systolic and diastolic blood pressure during our study in these two groups

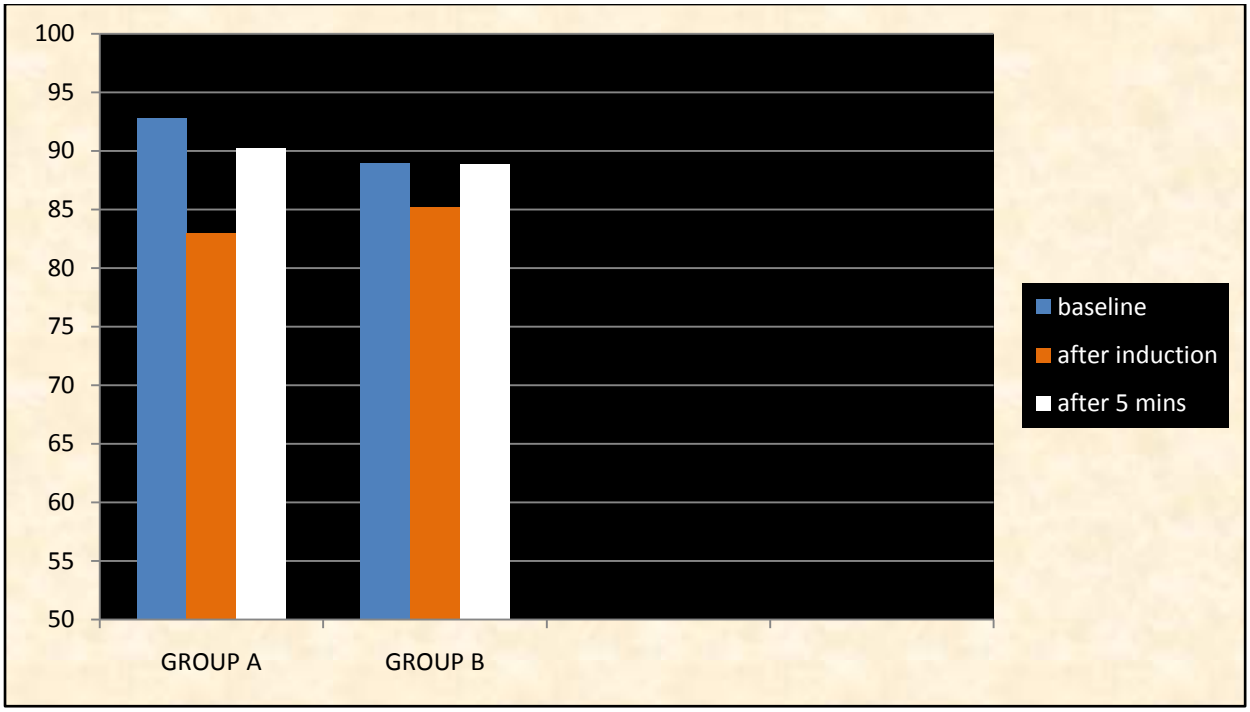

Fig 8: comparison MAP –baseline, after induction, and 5 min after induction

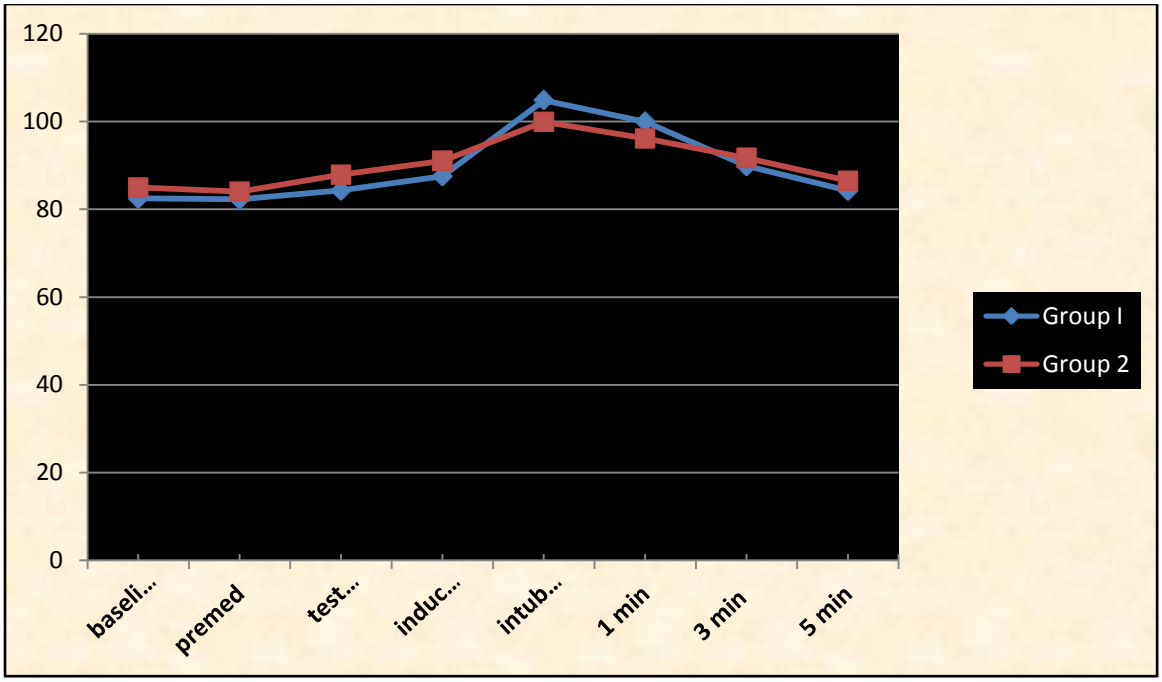

Fig 9: comparison of mean heart rates between the groups

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MASTER CHART

KETAMINE GROUP (GROUP- B) – DEMOGRAPHICS,HEART RATE,INTUBATION CONDITIONS

S NO.	NAME	AGE	SEX	HT	WT	BMI	ASA	MMS	HEART RATE								TIME	CL GRADE	POGO	INT-COND	INT SCORE
									BASAL	PREMED	KET	INDUCT	INT	1 MIN	3 MIN	5 MIN					TOTAL
1	DEVARAJ	52	M	156	50	20.5	1	1	94	88	92	102	112	98	90	86	148	II B	50	GOOD	7
2	FATHIMA	35	F	153	60	25.6	1	1	88	89	102	112	120	116	114	110	145	I	100	FAIR	5
3	THIGAMMA	60	F	157	60	24.3	1	1	76	68	84	102	110	104	100	88	93	I	100	EXCELLENT	8
4	ARAVIND	20	M	162	48	18.3	1	1	90	86	82	88	86	82	84	87	144	I	100	GOOD	7
5	BALAN	42	M	159	56	22.2	1	1	90	76	72	76	112	128	126	96	153	I	100	GOOD	7
6	MANOJ	23	M	166	60	21.8	1	1	80	70	78	76	90	86	80	84	88	II A	75	EXCELLENT	9
7	KUMAR	40	M	162	55	21.0	1	1	92	102	116	106	126	104	102	92	74	I	100	GOOD	7
8	MARIA	35	F	162	57	21.7	1	2	78	80	92	97	98	96	97	97	51	I	100	EXCELLENT	9
9	AMIRTHARAJ	22	M	165	75	27.5	1	2	97	104	97	92	124	102	104	108	86	II B	25	GOOD	7
10	KALPANA	26	F	146	45	21.1	1	2	104	102	106	96	116	102	96	90	69	I	100	EXCELLENT	8
11	MADHUBALA	20	F	155	50	20.8	1	2	90	76	82	86	88	84	92	90	78	I	100	EXCELLENT	9
12	VEERAMMAL	36	F	156	53	21.8	1	2	90	100	102	108	100	104	106	102	103	II A	100	FAIR	6
13	NAGAJYOTHI	45	F	152	53	22.9	1	2	85	77	89	92	105	110	98	82	77	II A	66	GOOD	7
14	DEVI	27	F	142	45	22.3	1	2	92	98	95	102	116	101	102	102	67	I	100	FAIR	5
15	JOHN JAYASEELAN	47	M	153	49	20.9	1	2	92	90	114	106	118	108	104	90	84	I	100	EXCELLENT	9
16	SELVAKUMARI	35	F	157	45	18.3	1	2	102	96	98	101	106	82	88	89	141	IIA	66	EXCELLENT	9
17	SUCHITRA	27	F	155	51	21.2	1	2	96	88	82	89	98	86	84	78	172	i	100	EXCELLENT	9
18	RAMA	45	F	158	55	22.0	2	2	82	78	88	84	92	90	86	84	93	IIA	66	EXCELLENT	9
19	VASANTHKUMAR	18	M	153	55	23.5	2	2	78	76	86	84	108	96	92	88	70	IIA	75	FAIR	4
20	TAMILSELVI	40	F	162	55	21.0	1	1	76	78	75	78	82	97	92	80	61	I	100	EXCELLENT	9
21	RAJESWARI	30	F	161	52	20.1	1	2	86	76	85	88	92	97	90	80	77	I	100	EXCELLENT	9
22	VIJAYA	38	F	157	55	22.3	1	2	85	86	90	96	112	120	104	90	145	IIA	75	EXCELLENT	9
23	SANGEETHA	50	F	152	50	21.6	1	1	84	88	88	105	110	88	82	80	79	I	100	EXCELLENT	9
24	SANJIVI	18	M	163	52	19.6	1	1	86	94	90	94	81	82	78	76	110	1	100	EXCELLENT	9
25	ANU	22	F	159	53	21.0	1	2	89	90	91	96	106	90	98	100	87	I	100	EXCELLENT	9
26	ASEENA BEGAM	30	F	154	45	19.0	1	2	89	90	92	97	96	90	90	94	62	1	100	EXCELLENT	9
27	CHITRA	34	F	165	57	20.9	1	2	83	85	92	90	104	110	100	105	57	1	100	EXCELLENT	9
28	KARTHIK	18	M	170	57	19.7	1	2	96	94	86	88	82	92	88	82	152	1	100	EXCELLENT	9
29	MALARKODI	18	F	153	45	19.2	1	2	85	89	102	96	104	102	86	89	64	1	100	GOOD	6
30	RAJESWARI	30	F	165	62	22.8	1	2	84	79	79	76	86	84	76	67	77	1	100	EXCELLENT	9

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31	MUTHULAKSHMI	24	F	163	59	22.2	2	2	72	70	82	112	106	100	90	89	60	1	100	EXCELLENT	9
32	REKHA	34	F	152	50	21.6	1	2	72	76	84	100	106	104	82	83	51	IIB	35	GOOD	6
33	SHANTHI	40	F	167	60	21.5	1	2	69	70	86	86	102	108	88	89	59	IIA	75	GOOD	7
34	PRAKASH	19	M	172	63	21.3	1	1	68	59	63	66	102	86	80	66	57	I	100	EXCELLENT	9
35	SUBBULAKSHMI	19	F	149	42	18.9	1	1	89	85	86	87	92	102	88	88	126	IIA	75	EXCELLENT	9
36	SARAVANAN	19	M	171	60	20.5	1	2	72	76	84	76	79	92	86	78	93	I	100	EXCELLENT	9
37	SURESH	35	M	183	70	20.9	1	1	82	82	84	78	76	82	78	62	82	1	100	GOOD	5
38	AIYASH	45	M	164	54	20.1	1	2	82	88	82	96	98	92	97	87	84	1	100	EXCELLENT	9
39	MADAVAN	40	M	179	69	21.5	1	2	78	80	64	66	63	66	72	69	130	IIA	66	FAIR	5
40	PARANTHAMAN	20	M	165	60	22.0	1	3	76	82	74	72	92	82	78	63	90	IIA	66	EXCELLENT	9

KETAMINE GROUP (GROUP- B) –HEMODYNAMICS

S NO.	NAME	SYSTOLIC BLOOD PRESSURE								DIASTOLIC BLLOD PRESSURE								MEAN ARTERIAL PRESSURE							
		BASAL	PREMED	KET	INDUCT	INT	1 MIN	3 MIN	5 MIN	BASAL	PREMED	KET	INDUCT	INT	1 MIN	3 MIN	5 MIN	BASAL	PREMED	KET	INDUCT	INT	1 MIN	3 MIN	5 MIN
1	DEVARAJ	150	140	146	90	133	126	130	140	78	80	78	60	84	72	70	84	102	100	101	70	100	90	90	103
2	FATHIMA	126	120	140	130	146	152	146	140	70	70	84	78	84	78	80	78	89	87	103	95	105	103	102	99
3	THIGAMMA	130	118	126	106	110	116	132	136	72	70	70	74	78	84	78	74	91	86	89	85	89	95	96	95
4	ARAVIND	110	102	114	126	156	140	146	142	76	64	72	74	84	78	76	78	87	77	86	91	108	99	99	99
5	BALAN	130	124	120	106	150	144	146	124	72	60	72	78	104	98	100	78	91	81	88	87	119	113	115	93
6	MANOJ	116	108	110	112	116	108	110	110	74	60	64	60	80	88	70	68	88	76	79	77	92	95	83	82
7	KUMAR	130	124	144	130	164	140	146	130	72	76	70	70	110	102	88	80	91	92	95	90	128	115	107	97
8	MARIA	127	126	130	105	104	105	110	100	80	70	76	60	76	69	70	52	96	89	94	75	85	81	83	68
9	AMIRTHARAJ	138	140	134	140	152	144	136	114	78	85	89	78	80	70	72	60	98	103	104	99	104	95	93	78
10	KALPANA	120	116	118	104	140	108	116	118	70	72	70	78	84	72	72	70	87	87	86	87	103	84	87	86
11	MADHUBALA	104	108	110	112	110	108	96	96	72	72	76	76	70	76	60	62	83	84	87	88	83	87	72	73
12	VEERAMMAL	108	104	106	91	98	96	92	97	72	70	72	64	62	64	58	65	84	81	83	73	74	75	69	76
13	NAGAJYOTHI	120	118	119	108	150	139	120	108	70	76	72	72	104	99	86	76	87	90	88	84	119	112	97	87
14	DEVI	104	102	106	92	134	110	98	102	76	76	70	56	74	70	64	76	85	85	82	68	94	83	75	85
15	JOHN JAYASEELAN	130	126	130	132	162	142	132	126	70	74	84	78	110	94	76	74	90	91	99	96	127	110	95	91
16	SELVAKUMARI	120	104	104	126	142	140	126	105	78	78	84	70	104	98	70	76	92	87	91	89	117	112	89	86
17	SUCHITRA	120	104	110	104	132	105	108	104	72	80	78	76	84	76	78	72	88	88	89	85	100	86	88	83
18	RAMA	104	108	130	138	142	138	126	124	76	72	76	99	102	100	96	90	85	84	94	112	115	113	106	101
19	VASANTHKUMAR	104	108	112	110	124	108	96	92	78	84	70	76	70	70	70	76	87	92	84	87	88	83	79	81

MASTER CHART

20	TAMILSELVI	120	124	120	112	108	120	124	122	70	82	80	84	84	82	80	86	87	96	93	93	92	95	95	98
21	RAJESWARI	110	114	110	112	108	110	104	102	70	72	70	72	70	70	70	76	83	86	83	85	83	83	81	85
22	VIJAYA	110	114	112	96	142	130	120	108	72	78	70	54	108	90	74	70	85	90	84	68	119	103	89	83
23	SANGEETHA	98	96	94	88	118	96	90	92	72	70	72	56	88	76	70	76	81	79	79	67	98	83	77	81
24	SANJIVI	102	108	102	108	122	124	128	130	78	84	76	70	80	78	76	84	86	92	85	83	94	93	93	99
25	ANU	108	122	124	129	128	124	130	140	70	76	70	87	78	78	76	95	83	91	88	101	95	93	94	110
26	ASEENA BEGAM	96	98	110	102	114	108	88	89	56	60	74	78	72	76	70	72	69	73	86	86	86	87	76	78
27	CHITRA	148	120	127	128	126	136	122	130	100	80	84	87	85	88	84	82	116	93	98	101	99	104	97	98
28	KARTHIK	114	110	108	110	124	148	118	116	86	76	74	70	86	84	74	73	95	87	85	83	99	105	89	87
29	MALARKODI	118	126	123	126	123	140	136	120	70	76	78	84	86	78	78	76	86	93	93	98	98	99	97	91
30	RAJESWARI	110	112	124	102	110	114	106	93	70	72	76	70	72	70	70	62	83	85	92	81	85	85	82	72
31	MUTHULAKSHMI	126	120	136	109	122	146	96	99	72	56	72	70	90	84	54	58	90	77	93	83	101	105	68	72
32	REKHA	126	120	134	96	114	119	120	119	78	72	72	58	80	90	70	83	94	88	93	71	91	100	87	95
33	SHANTHI	104	110	126	114	144	140	126	128	76	72	72	70	80	88	80	72	85	85	90	85	101	105	95	91
34	PRAKASH	120	110	114	78	110	104	106	100	80	70	72	50	72	70	72	54	93	83	86	59	85	81	83	69
35	SUBBULAKSHMI	108	104	110	116	130	126	124	114	78	76	72	72	70	78	72	78	88	85	85	87	90	94	89	90
36	SARAVANAN	110	116	118	110	143	130	124	120	76	74	76	70	86	88	92	90	87	88	90	83	105	102	103	100
37	SURESH	142	140	138	126	142	138	110	118	76	72	70	72	108	72	70	74	98	95	93	90	119	94	83	89
38	AIYASH	138	140	142	140	148	146	140	143	76	84	78	70	92	84	78	98	97	103	99	93	111	105	99	113
39	MADAVAN	140	110	110	116	113	110	115	116	84	70	72	78	72	70	76	77	103	83	85	91	86	83	89	90
40	PARANTHAMAN	122	124	120	110	140	123	120	130	70	70	76	70	102	76	72	90	87	88	91	83	115	92	88	103

MASTER CHART

SALINE GROUP (GROUP- A) – DEMOGRAPHICS,HEART RATE,INTUBATION CONDITIONS

S NO.	NAME	AGE	SEX	HT	WT	BMI	ASA	MMS	HEART RATE								TIME	CL GRADE	POGO	INT-COND	INT SCORE
									BASAL	PREMED	SALINE	INDUCT	INT	1 MIN	3 MIN	5 MIN					TOTAL
1	SUGANTHI	41	F	157	55	22.3	2	2	82	89	88	68	78	89	76	68	60	I	100	GOOD	7
2	SELVI	38	F	153	50	21.4	2	2	84	92	90	93	120	112	108	99	225	I	100	FAIR	5
3	GOWRI	34	F	165	65	23.9	1	1	87	80	82	96	112	102	98	90	147	I	100	EXCELLENT	9
4	RAJA	36	M	151	45	19.7	1	1	74	76	72	123	120	104	106	96	147	I	100	EXCELLENT	9
5	REKHA	18	F	153	45	19.2	1	2	74	76	74	120	126	108	94	88	83	IIA	50	FAIR	5
6	PAVITHRA	18	F	147	40	18.5	1	1	78	86	82	86	106	102	92	86	92	I	100	GOOD	7
7	CHINNAPONNU	40	F	154	50	21.1	2	2	84	84	86	120	126	108	84	72	84	IIA	50	GOOD	7
8	LOURD MARY	40	F	175	85	27.8	1	2	98	86	82	86	106	102	92	86	77	I	100	EXCELLENT	9
9	CHANDRAIAH	50	M	167	60	21.5	1	1	86	90	92	88	110	106	84	82	170	I	100	EXCELLENT	9
10	KOTHANDAN	52	M	163	56	21.1	1	2	68	72	70	86	82	84	82	78	149	IIA	66	EXCELLENT	8
11	RAJESWARI	40	F	164	61	22.7	1	2	106	98	88	86	92	88	86	84	170	I	100	EXCELLENT	9
12	KAMATCHI	50	F	179	75	23.4	1	1	68	66	62	66	72	92	62	61	63	IIA	66	GOOD	7
13	KALAIARASI	40	F	171	65	22.2	1	3	64	82	84	75	92	96	76	76	65	I	100	EXCELLENT	9
14	AMALU	30	F	162	57	21.7	1	1	81	79	78	82	80	92	76	74	77	IIB	33	EXCELLENT	9
15	DEVI	50	F	149	50	22.5	1	2	96	88	86	76	114	96	84	80	198	IIB	33	EXCELLENT	9
16	SAKTHIVEL	31	M	162	55	21.0	1	2	89	92	86	80	116	92	88	84	96	IIA	66	FAIR	5
17	SEETHA	26	F	155	52	21.6	1	2	54	82	78	82	96	84	86	87	127	I	100	EXCELLENT	9
18	KALAISELVI	35	F	146	45	21.1	1	1	92	96	94	86	130	104	104	90	210	I	100	EXCELLENT	9
19	SURESH	20	M	162	58	22.1	1	1	76	72	86	89	102	76	78	74	113	IIA	66	FAIR	5
20	VINOTHKUMAR	28	M	173	70	23.4	1	2	84	78	76	86	122	126	108	92	108	I	100	EXCELLENT	9
21	RAHUL	26	M	164	55	20.4	1	1	77	82	84	78	70	76	82	84	158	I	100	EXCELLENT	9
22	AMUDHA	57	F	160	57	22.3	1	2	86	92	96	89	102	86	88	84	213	I	100	EXCELLENT	9
23	SHEELA	35	F	155	52	21.6	1	2	92	101	105	96	113	105	103	98	92	IIIA	0	FAIR	5
24	POUNAMMAL	45	F	153	52	22.2	1	2	87	86	86	73	106	102	78	75	222	IIB	66	FAIR	5
25	SENTHILNATHAN	40	M	182	72	21.7	1	1	92	90	102	106	128	108	96	80	143	I	100	EXCELLENT	9
26	SASHI	30	F	152	46	19.9	1	1	86	88	92	82	121	113	98	96	103	IIA	66	GOOD	7
27	RAJENDIRAN	44	M	169	69	24.2	1	2	71	69	75	71	92	97	91	85	159	I	100	EXCELLENT	8
28	MUTHU	37	M	178	80	25.2	1	2	98	82	90	110	108	116	98	90	92	IIB	33	EXCELLENT	8

MASTER CHART

29	NIROSHA	21	F	152	55	23.8	1	1	86	82	90	85	110	111	108	84	132	I	100	GOOD	7
30	MUTHUMANIKANDAN	20	M	159	52	20.6	1	1	86	88	84	92	86	96	89	80	170	i	100	EXCELLENT	9
31	GOKUL	20	M	157	51	20.7	1	1	92	89	104	89	106	110	108	105	141	I	100	EXCELLENT	9
32	MURALI	19	M	163	58	21.8	1	2	60	58	62	49	82	78	62	61	106	I	100	EXCELLENT	9
33	SOWMIYA	16	F	155	48	20.0	1	1	82	78	104	96	126	110	81	86	210	I	100	EXCELLENT	9
34	MAHENDRAN	26	M	156	60	24.7	1	1	88	86	88	84	124	116	94	80	91	IIA	66	GOOD	7
35	ARCHANA	18	F	145	47	22.4	1	2	85	87	92	102	110	108	104	106	72	IIA	66	GOOD	7
36	JAYALAKSHMI	18	F	153	50	21.4	1	3	82	78	84	86	92	84	78	75	143	I	100	EXCELLENT	9
37	SANDHIYA	23	F	156	58	23.8	1	1	82	78	80	76	82	88	87	82	124	IIA	50	GOOD	7
38	MAHESH	19	M	167	58	20.8	1	1	82	68	74	86	116	110	81	86	110	I	100	FAIR	5
39	SUBRAMANI	36	M	154	60	25.3	1	1	74	65	66	91	95	95	94	92	50	I	100	GOOD	6
40	ELAYVENDHAN	28	M	173	70	23.4	1	2	84	78	76	86	122	126	108	92	108	I	100	FAIR	5

SALINE GROUP (GROUP- A) – HEMODYNAMICS

S NO.	NAME	SYSTOLIC BLOOD PRESSURE								DIASTOLIC BLLOD PRESSURE								MEAN ARTERIAL PRESSURE							
		BASAL	PREMED	SALINE	INDUCT	INT	1 MIN	3 MIN	5 MIN	BASAL	PREMED	SALINE	INDUCT	INT	1 MIN	3 MIN	5 MIN	BASAL	PREMED	SALINE	INDUCT	INT	1 MIN	3 MIN	5 MIN
1	SUGANTHI	140	129	132	139	136	140	120	110	80	64	76	100	110	102	86	72	100	86	95	113	119	115	97	85
2	SELVI	108	107	115	86	88	84	96	111	85	85	81	50	56	52	68	82	93	92	92	62	67	63	77	92
3	GOWRI	110	114	118	96	130	130	124	126	72	76	70	64	76	70	78	72	85	89	86	75	94	90	93	90
4	RAJA	114	110	114	85	132	116	110	118	87	85	90	69	78	72	78	84	96	93	98	74	96	87	89	95
5	REKHA	116	114	110	86	164	126	124	122	70	72	72	54	70	72	70	70	85	86	85	65	101	90	88	87
6	PAVITHRA	150	146	147	132	138	146	140	140	80	74	78	70	90	84	80	80	103	98	101	91	106	105	100	100
7	CHINNAPONNU	116	104	108	90	144	126	124	122	70	72	72	54	70	72	70	70	85	83	84	66	95	90	88	87
8	LOURD MARY	150	146	152	132	150	170	146	140	80	74	68	73	100	104	74	70	103	98	96	93	117	126	98	93
9	CHANDRAIAH	116	118	110	104	150	148	130	128	70	72	80	76	114	108	90	84	85	87	90	85	126	121	103	99
10	KOTHANDAN	120	156	150	116	124	123	120	125	99	90	86	78	72	76	80	90	106	112	107	91	89	92	93	102
11	RAJESWARI	124	146	126	124	126	134	130	110	86	92	78	72	90	88	80	72	99	110	94	89	102	103	97	85
12	KAMATCHI	158	140	142	124	120	103	88	103	64	76	78	72	76	70	64	76	95	97	99	89	91	81	72	85
13	KALAIARASI	106	116	120	108	122	124	123	126	84	72	72	76	84	78	80	91	91	87	88	87	97	93	94	103

MASTER CHART

14	AMALU	124	108	104	102	96	110	124	130	76	68	72	70	54	70	70	72	92	81	83	81	68	83	88	91
15	DEVI	160	140	142	126	160	140	139	132	88	84	90	72	102	80	78	76	112	103	107	90	121	100	98	95
16	SAKTHIVEL	130	128	128	96	118	164	124	130	84	78	76	54	76	84	78	78	99	95	93	68	90	111	93	95
17	SEETHA	110	121	116	89	112	110	112	101	72	76	76	64	76	78	76	64	85	91	89	72	88	89	88	76
18	KALAISELVI	106	110	104	98	150	144	156	143	72	70	72	64	98	110	98	70	83	83	83	75	115	121	117	94
19	SURESH	124	110	115	80	122	98	106	103	76	72	70	56	84	54	73	70	92	85	85	64	97	69	84	81
20	VINOTHKUMAR	128	126	124	106	158	102	96	98	72	76	70	70	96	76	54	50	91	93	88	82	117	85	68	66
21	RAHUL	126	116	110	117	136	110	108	112	76	80	82	84	70	76	82	80	93	92	91	95	92	87	91	91
22	AMUDHA	124	110	116	89	122	98	106	103	76	72	70	56	84	54	73	70	92	85	85	67	97	69	84	81
23	SHEELA	116	118	126	128	140	114	110	112	80	76	74	70	84	99	80	78	92	90	91	89	103	104	90	89
24	POUNAMMAL	105	108	106	91	150	140	128	124	82	79	80	62	72	102	70	76	90	89	89	72	98	115	89	92
25	SENTHILNATHAN	124	126	124	120	140	120	116	124	92	78	70	76	84	70	76	72	103	94	88	91	103	87	89	89
26	SASHI	126	120	114	109	144	117	106	110	72	70	70	58	102	81	78	70	90	87	85	75	116	93	87	83
27	RAJENDIRAN	135	128	115	114	148	144	127	100	78	70	88	80	99	110	99	77	97	89	97	91	115	121	108	85
28	MUTHU	130	136	142	96	140	148	136	138	78	84	86	64	110	98	96	96	95	101	105	75	120	115	109	110
29	NIROSHA	108	110	114	98	138	144	120	115	72	76	72	64	82	98	70	72	84	87	86	75	101	113	87	86
30	MUTHUMANIKANDAN	124	126	122	116	124	126	116	127	72	78	76	78	70	72	82	87	89	94	91	91	88	90	93	100
31	GOKUL	126	128	124	86	144	138	130	145	70	72	80	64	80	78	70	72	89	91	95	71	101	98	90	96
32	MURALI	126	123	122	126	144	136	123	122	78	74	70	70	110	98	76	72	94	90	87	89	121	111	92	89
33	SOWMIYA	104	106	116	110	124	116	110	108	72	76	78	70	90	72	72	70	83	86	91	83	101	87	85	83
34	MAHENDRAN	140	136	146	140	140	136	146	130	98	90	97	84	90	88	76	78	112	105	113	103	107	104	99	95
35	ARCHANA	110	116	110	108	153	132	136	140	72	72	72	84	110	107	108	102	85	87	85	92	124	115	117	115
36	JAYALAKSHMI	108	112	110	116	134	141	106	112	72	70	76	72	84	82	72	69	84	84	87	87	101	102	83	83
37	SANDHIYA	110	116	112	108	138	120	120	114	72	70	70	64	78	62	68	72	85	85	84	79	98	81	85	86
38	MAHESH	104	106	118	112	114	116	107	110	62	72	76	90	70	72	72	68	76	83	90	97	85	87	84	82
39	SUBRAMANI	148	134	130	128	130	130	128	143	89	92	90	92	100	107	90	90	109	106	103	104	110	115	103	108
40	ELAYVENDHAN	128	126	124	106	158	102	96	98	72	76	70	70	96	76	54	50	91	93	88	82	117	85	68	66

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. D. Naveen Kumar
PG in M.D.Anaesthesiology
Madras Medical College, Chennai -3

Dear Dr.D.Naveen Kumar,

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled " The effect of ketamine on the onset time and the intubating conditions of rocuronium bromide" No.20112012.

The following members of Ethics Committee were present in the meeting held on 01.11.2012 conducted at Madras Medical College, Chennai -3.

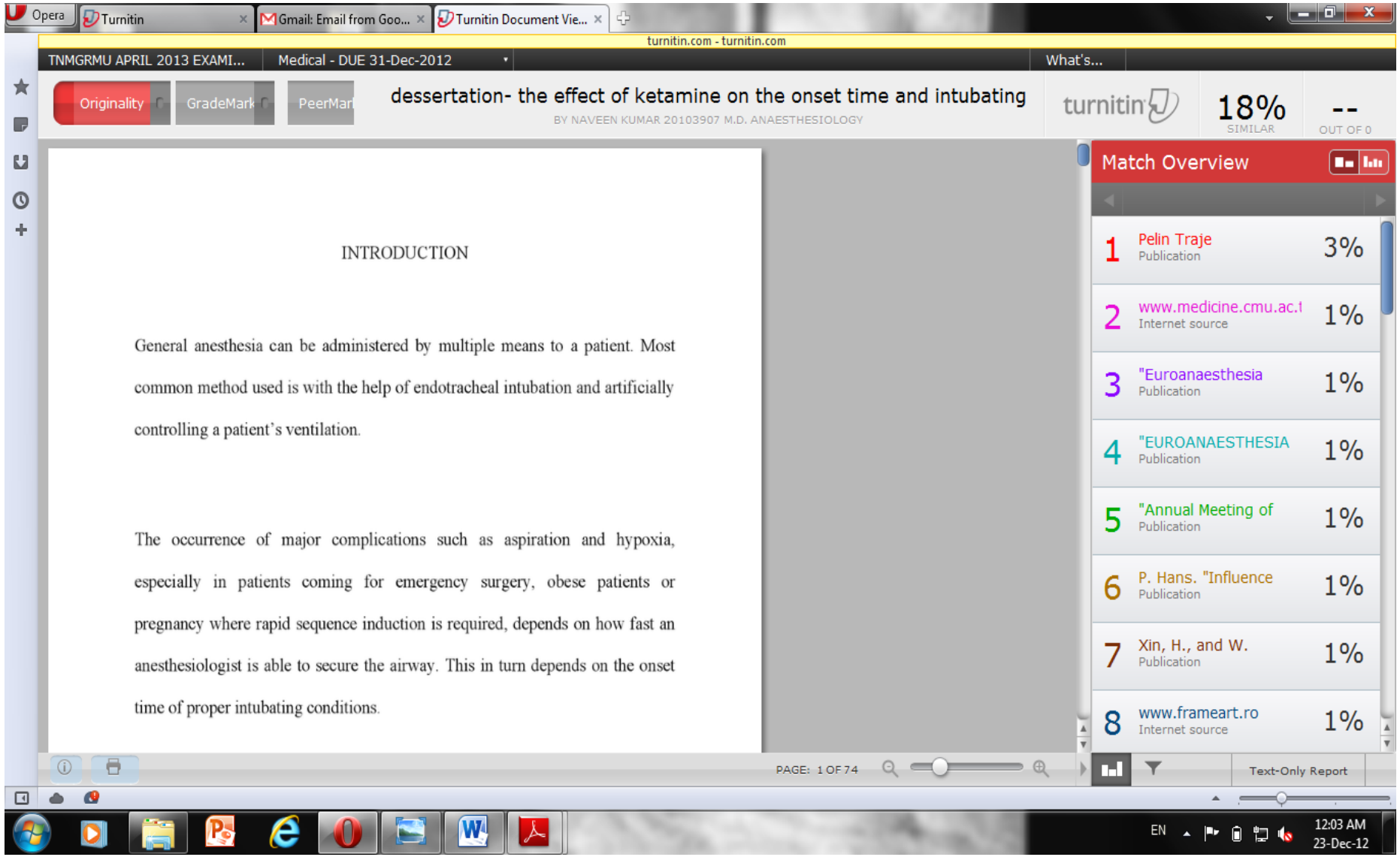
1.	Prof. R. Nandhini MD Director, Instt. of Pharmacology ,MMC, Ch-3	-- Member Secretary
2.	Prof. Reghu MD Director , Inst. Of Internal Medicine, MMC, Ch-3	-- Member
3.	Prof. Shyamraj MD Director i/c , Instt. of Biochemistry , MMC, Ch-3	-- Member
4.	Prof. P. Karkuzhali. MD Prof., Instt. of Pathology, MMC, Ch-3	-- Member
5.	Prof. G.Muralidharan MS Prof of Surgery, MMC, Ch-3	-- Member
6.	Thiru. S. Govindsamy. BA,BL	-- Lawyer

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

RNandini 19/11/12
Member Secretary, Ethics Committee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INTRODUCTION
General anesthesia can be administered by multiple means to a patient. Most common method used is with the help of endotracheal intubation and artificially controlling a patient's ventilation.
The occurrence of major complications such as aspiration and hypoxia, especially in patients coming for emergency surgery, obese patients or pregnancy where rapid sequence induction is required, depends on how fast an anesthesiologist is able to secure the airway. This in turn depends on the onset time of proper intubating conditions.
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PROFORMA

NAME: **AGE:** **SEX:** **I.P. NO.**

DIAGNOSIS: **WT:** **MMS CLASS:**

SURGERY PERFORMED: **TEST DRUG:**

HEMODYNAMICS:

	HR	BP
BASELINE		
AFTER PREMEDICATION		
AFTER TEST DRUG		
AFTER INDUCTION		
AFTER INTUBATION		
1 MIN		
3 MIN		
5 MIN		

TIME:

TEST DRUG	**SEC**
TIME FOR DISAPPEARANCE OF TI IN TOF OR STS	**SEC**

INTUBATING CONDITIONS:

- **CORMACK LEHANNE GRADE –**
- **POGO SCORE –**
- **GRADING OF INTUBAING CONDITIONS BY COOPER, ET AL**

SCORE	JAW RELAXATION	VOCAL CORDS	RESPONSE TO INTUBATION
0	POOR	CLOSED	SEVERE COUGHING/BUCKING
1	MINIMAL	CLOSING	MILD COUGHING
2	MODERATE	MOVING SLIGHTLY	DIAPHRAGMATIC MOVEMENT
3	GOOD	OPEN	NONE
TOTAL SCORE		INTUBATING CONDITIONS	

Score 8-9 = Excellent, 6-7 = Good, 3-5 = Fair, 0-2 =Poor

INFORMATION TO PARTICIPANTS

Investigator **:** Dr.Naveen Kumar .D

Name of the Participant:

Title

The Effect Of Ketamine On The Onset Time And The Intubating Conditions Of Rocuronium Bromide

You are invited to take part in this research study. We have got approval from the IEC. Your are asked to participate because you satisfy the eligibility criteria .We want to study the effect of ketamine on the intubating conditions of propofol rocuronium induction.

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What is the Purpose of the Research:

This is done to study the effect of ketamine on the intubating conditions and the onset time of propofol rocuronium induction on elective surgeries. The onset time of a neuromuscular blocking drug is an important factor in determining the speed and ease with which the trachea can be intubated especially for rapid sequence induction for which suxamethonium is used which can cause complications in certain conditions like hyperkalemia,cholinesterase deficiency,burns,penetrating eye injury,allergic reactions. Nevertheless, speed of onset of rocuronium is dose-dependent and high doses are associated with a long duration of action. We hypothesized that the addition of ketamine to a propofol induction would improve intubating conditions after rocuronium while shortening the onset time of neuromuscular block thus avoiding the undue side effects of suxamethonium and also high dose of rocuronium.

The Study Design:

to study the effects of ketamine on the intubating conditions of propofol rocuronium induction on elective surgeries. You will be allocated into one of the two groups after getting informed consent.after premedicating with drugs to reduce your anxiety we will give drugs to anesthetise you and get trachea intubated.various parameters such as heart rate, bp etc will be measured.

Benefits:

With ketamine , there would be better intubating conditions with shorter onset time and shorter duration of muscle relaxant as it would be useful in short duration surgeries. It would be useful in avoiding unwanted side effects of suxamethonium. The fall in BP that can occur following a normal induction is offset by use ketamine

Discomforts and risks:

Raise in heart rate and blood pressure can occur..If heart rate increases then we giveinj xylocard or inj.esmolol. there is a possibility of emergence delirium in few patients which can be readily treated with midazolam. There is risk of cannot ventilate and cannot intubate situation which can occur in 0.1% of population where emergency airway may be placed like laryngeal mask airway, tracheostomy or cricothyroidotomy

This intervention has been shown to be well tolerated as shown by previous studies. And if you do not want to participate you will have alternative of setting the standard treatment and your safety is our prime concern.

Time :
Date :
Place :

Signature / Thumb Impression of Patient
Patient Name:

Signature of the Investigator : _____

Name of the Investigator : _____

PATIENT CONSENT FORM

Study title

the effect of ketamine on the onset time and the intubating conditions of rocuronium bromide

Study centre : Department of Anaesthesiology
Institute of Anesthesiology and critical care,
Madras Medical College
Chennai 600003

Participant name : Age: Sex: I.P.No:

I confirm that i have understood the purpose of procedure for the above study . i have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the pitfall in the procedure. I have been explained about the safety,advantage and disadvantage of the technique.

I understand that my participation in the study is voluntary and that i am free to withdraw at anytime without giving any reason.

I understand that investigator ,regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in relation to it, even if i withdraw from the study . I understand that my identity will not be revealed in any information released to third parties or published , unless as required under the law . I agree not to restrict the use of any data or results that arise from the study .

Time:

Date: signature / thumb impression of patient

Place: patient name:

Signature of the investigator:

Name of the investigator: